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OF THE

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ITHACA, N. Y.

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Communications relating to membership and matters pertaining to the American Veterinary Medical Association itself should be addressed to Secretary C. M. Haring, University of California, Berkeley, California. Matters pertaining to the Journal should be sent to Ithaca, N. Y.

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## A FORECAST

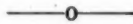
As we are assembling our material for this number, word comes to us of some of the results of the efforts of chairman Merillat and Higgins in building up a program, which shall have interesting and instructional features for the profession at large and even extend beyond the profession to those interested in stock breeding, sanitation and public health as it relates to the domesticated animals.

At this time we can announce that there will be a symposium on contagious abortion; a half-day section on horse breeding and remounts; papers by men of national prominence on surgery, lameness, pharmacology, local anesthesia, animal dentistry, forage poisoning, cholera, canine distemper, shipping fever, calf scours and others, to be announced later. In addition, there will be reports from the various committees which should be of as much general interest to the profession as the program. The report of the legislative committee should be of especial interest this year. Of no less interest should be the reports of the committees on intelligence and education, reorganization, diseases and various others.

Although this is but a brief outline of the good things that may

be expected, it strikes us that it is sufficient to stimulate a large interest in the membership and should insure a large attendance. It should also stimulate much interest among those who are not members and induce many of them to join the association. We hope, at least, that many of them will attend the meetings, at which they will be cordially welcome, and judge for themselves if they cannot further the best interests of the profession, as well as their own efficiency by working with the association.

Mark the date, August 21st, and remember that on that date all roads lead to Detroit. If it has not already been done, make your reservations at the official headquarters, the Statler Hotel, or at some of the other hotels conveniently located, as published in the June number of the JOURNAL. P. A. F.



### IMPAIREDNESS OR PREPAREDNESS

Until recently we have had a notable example of impairedness in the veterinary branch of the army, because of the refusal of the government to grant the veterinarians rank and standing. A jolt is sometimes necessary to arouse one from a lethargy in which he has allowed himself to sink. Our country has untold treasure without adequate protection and has seemed strangely indifferent to its preservation. The possibility of being drawn into the present whirlpool of war, however unwillingly, seems to have aroused some from their lethargy. This possibility has been the jolt which has caused thoughtful ones to take an inventory of our resources and to consider our readiness to repel an invasion, if such were offered. Under changed conditions of warfare, the oceans no longer offer the same protection as in years gone by. A nation contemplating war does not publish its plans in advance. War may come unexpectedly and there is but little time for preparation,—if it has not already been made. There are not many nations which have reached that degree of Christian humility that when smitten upon one cheek they will turn the other cheek also, and will welcome a foreign power as an overlord. Considerations of this character have demanded and caused the passage of an army bill with which has been carried a more adequate recognition of veterinary service.

The present war, among other things, has emphasized the necessity for an adequate and competent veterinary branch. It has



done more. It has shown that it is of the utmost importance to have an abundant supply of horse material available in time of need and that this material should be of the highest quality. With Russia as the only exception, the United States is the greatest producer of horses in the world. It has been estimated that out of the 24,000,000 horses in this country, only about 224,000 are of the type suitable for military purposes. Something less than one per cent of what we have are suitable for our use in case of necessity. Even this small percentage has been depleted by export during the last two years. It is folly to assume that foreign agents have accepted for use of their governments, anything that would pass as a horse. There is no question that we have the quantity, but the quality of the horses is the serious problem. The significance of our present impairedness in this direction is emphasized by the fact that during one year of the Civil War the government purchased 221,000 mounts.

Have we adequate army veterinary service to properly care for this number and the many more that would be required in case of another and perhaps greater war? The effort to establish a veterinary reserve corps and the voluntary response of a goodly number of patriotic veterinarians to serve in this way is most gratifying. It is hoped that enough momentum may be gained to compensate, to some extent, for the inertia of the past.

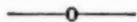
It has been stated that some of the newer and important agents used in the present struggle were invented in this country and offered to our government, which was not interested, and then disposed of abroad where they have been perfected and lost to our exclusive use. Others have prepared from our inventions while we have generously impaired our own efficiency. This procedure should not extend to the type of horse required for military use. The scrub should be allowed to die out and only the best of the various types propagated for future use. Foreign governments have spent thousands where we have spent hundreds, or less, for this purpose. Our government will find it a paying investment to encourage and stimulate stock breeders to interest themselves in the better types only.

Associated with the horse are roads. Again, the present conflict has emphasized the necessity for good road-beds. Railway facilities have been shown to be inadequate, and for greater mobil-

ity of troops and quick dispatch to isolated areas, good roads have been imperative. It has been possible, by their presence, to snatch victory from defeat in many instances. It is not to be inferred that the mobility and quick dispatch of troops is dependent upon the horse so much as upon the use of motor-cars, but the good roads are advantageous for horses as well as motors, and the horse has the further advantage that he can go where the car cannot.

Good horses and good roads are imperative in war; they are quite as essential in peace, and both concern the veterinarian. It is difficult to see how the most extreme pacifist can object to these forms of preparedness. It is not a question of preparedness alone, but a question of thrift against waste; of efficiency against inefficiency. The one promises a useless sacrifice of life and treasure; the other safeguards them. Money is cheaper than life. Let us woo peace even if it requires super-preparedness. Our country is concerned with the divine rights of humanity. We believe that the principles upon which this government was established are worth living for in peace and dying for in war.

P. A. F.



### IMPROVEMENT IN THE PREPARATION OF ANTI-HOG CHOLERA SERUM

In an article by Drs. Dorset and Henley of the Biochemic Division of the Bureau of Animal Industry, appearing in a recent number of the *Journal of Agricultural Research*, was described the production of a clear and sterilized anti-hog cholera serum. It is certainly gratifying that with the simple procedure as devised by these investigators it will now be possible to prepare a product which will in all respects prove more satisfactory and also more scientific.

There is no doubt that hog cholera serum has probably been the most important biological product produced in recent years, and its effectiveness in hog cholera caused by the filterable virus is no longer questioned.

Hog cholera serum, however, in its former preparation represented a crude product, frequently contaminated with various kinds of germs. This shortcoming was mainly responsible for unjust criticism as to the efficiency and potency of the product, and

veterinarians no doubt have also very frequently despaired on account of bad results which followed its administration. Septic and pyemic conditions not infrequently followed the injection of contaminated serum, and we are not at all certain whether the increased number of cases of polyarthritis and multiple abscesses in hams observed in the abattoirs might not have been directly due to contaminated serum.

The simplicity of the technique described by Dr. Dorset as required in the production of his clear and sterilized anti-hog cholera serum will no doubt revolutionize this product, and it is hoped that veterinarians in the near future will be in a position to avail themselves of this improved and highly important preparation.

In the clear serum all of the anti-bodies of the blood are contained, and only the inert blood corpuscles are eliminated, which is another advantage, since it no doubt hastens its absorption. The possibility of heating the same to 60°C., or passing it through a Berkefeld filter should also appeal to the users of this product, since through filtration the obnoxious contaminations may be removed, and with the heating the danger of the possibility of a serum contaminated with foot-and-mouth disease virus is avoided.

Drs. Dorset and Henley deserve the hearty congratulations of the veterinary profession for their new achievement, and it is hoped the method will soon be adopted on a large scale by commercial producers of serum.

A. E.

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### U. S. VETERINARY INSPECTORS

It is quite generally recognized that the veterinarians in the federal meat inspection are underpaid. This is not due to any fault of the Bureau of Animal Industry but rather to the limitations of the appropriations available for its use. That the work involved in this branch of the service is of great importance cannot be successfully controverted. It is therefore necessary that the inspectors must be of a high grade. They must be graduates of reputable veterinary colleges and must have spent considerable time and money in acquiring a sufficient education for the purpose. To hold such men, when ordinary practice usually brings in greater incomes, it is essential that adequate salaries should be paid—or else the service loses.

An effort is being made to place the matter on a more stable and just foundation through the Lobeck bill, which provides for a salary of \$1400 on entering the service and an annual increase of \$100 thereafter until the salary shall amount to \$2400 per annum. It is gratifying to report, at this writing, that the committee has voted to make a favorable report on this bill to the House and that within a few days it will be placed upon the House calendar. It will also be introduced into the Senate at an early date. It is obvious if the government is to retain the best men in this branch of its service, it must pay adequate salaries. The Bureau inspectors and clerks, as well as the profession at large, should feel under great obligation to Mr. Lobeck for the interest and energy he has shown in promoting this act of justice.

P. A. F.

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### IMMATURE MEAT

Practically all of the legislation against the use of immature meat has been centered around veal. We have heard little or nothing about lambs or pigs; yet it is reasonable to infer that such meat is just as dangerous from one source as another. The calf seems to have been the recipient of much class or special legislation. There have undoubtedly been more attempts to use immature calves than of the others. Yet the fundamental principle is the same in all and legislation should be impartial in a matter of this character.

If there has been one reason more emphatic than any other in hampering efficient inspection in this direction and more illogical in principle, it has been legislation specifying the age limit. There has been no attempt at coordinating this time limit between federal, state and municipal authorities. This has resulted in a most chaotic state of affairs and presented many opportunities for controversy. In some sections a veal is legally immature until it is three weeks of age; in others, it must be at least six weeks old. There is a difference of 100% in this age limit and this range seems unnecessarily wide. Just what is the determining factor, aside from the age, is not clear. That age ordinarily confers more maturity is incontrovertible, but it does not determine that younger veal is unwholesome or harmful. In cases brought to court, the crises, in many instances, develops around the age limit. If the burden of proof is



placed upon the inspector, he must possess superhuman qualities, in the absence of definite information, to swear to the exact age of the animal. Conditions of this character afford fertile opportunities for legal complications. These complications are not always creditable to the prosecutors. Opinion has been superior to evidence because until late years there has been no very direct evidence. A newspaper commenting on some of this evidence, stated its position as favoring the opinion of physicians who pronounced that bob veal was harmful if not actually poisonous, rather than to accept the evidence of harmlessness. It preferred prejudice to proof. There has been evidence for some time in the practices of foreign countries in consuming, what would be condemned in this country as immature meat, without apparent detriment to the health of the consumers.

The work of Berg of the Bureau of Animal Industry has shown that from a chemical standpoint the differences between immature veal and beef were negligible and from experiments in artificial digestion and feeding tests on cats that the physiological differences were insignificant. There has been previous confirmation of some of this work and there is a satisfaction in having direct evidence from a federal department in this matter rather than speculation.

To those properly trained it may not be especially difficult to detect either extreme as regards mature or immature meat, but the borderline between the two requires superhuman powers. To determine it by the age limit is entirely unreliable because of the physiologic differences that exist in each individual from the fetal stage onward. The criterion should be the quality of the meat; some immature meat may be unwholesome just as some mature meat may be unwholesome, and this can be determined by careful inspection. The statements that immature meat is deleterious to the health, if not actually poisonous, need modification. The same standards of inspection should apply to all meats—quality and wholesomeness. Without the complex question of age limit, there is less likelihood of litigation and, in some cases, possibly less unintentional perjury.

Age does not necessarily confer either health or wholesomeness. There are some qualities which may be recognized as more or less characteristic of immature meat, and these the Bureau of Animal Industry, since November, 1914, has recorded as follows: "Carcasses shall be considered too immature (and this refers to pigs, kids



and lambs, as well as calves) to produce wholesome meat if the meat has the appearance of being watersoaked, is loose, flabby, tears easily, and can be perforated with the fingers; or its color is grayish red; or good muscular development as a whole is lacking, especially noticeable on the upper shank of the leg, where small amounts of serous infiltrates or small edematous patches are sometimes present between the muscles; or the tissues which later develop as the fat capsule of the kidneys is edematous, dirty yellow or grayish red, tough and intermixed with islands of fat."

Experience may demonstrate the desirability of subtracting from or adding to these clues, but there is at least something more definite here than guesses at the age of the animal. We believe the Bureau has taken a progressive stand on a sound basis, and we cannot do more than commend it to the careful consideration of the legislators of the various states and municipalities as a wise example for them to follow. This is one of the questions where uniformity is possible and most desirable in eliminating present confusing conditions as to legal complications, waste and inefficiency.

P. A. F.

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## EUROPEAN CHRONICLES

*Bois, Jerome.*

**BACTERIOTHERAPY AND WOUNDS.** Last year in one of the visits that Prof. Lignieres of Buenos Ayres made to his native country, he presented a communication on this subject before the *Societe Centrale*. The application of bacteriology to the treatment of wounds was certainly a subject of interest which could not fail to draw attention inasmuch as the writer of the communication is an authority so well known.

"Today," said the learned professor, "there is no method of treatment of wounds, which would not be interesting, if one knows how to apply it according to the various cases that may exist." It is for that reason that the following observations made on bacteriotherapy are presented.

There are necrotic wounds, where the medicinal substances, including serum, reach the deeply injured points only with difficulty. For instance in the horse, those found in some cases of quittor, of poll evil or diseased withers. Could not a microbial in-

jection bring on or hasten recovery by a specific action in the very organism of the sick individual?

Indeed, when, in cartilaginous quittor, the fistulous tracts are cleaned with the ordinary methods, but without operation, and three or four subcutaneous injections of dead cultures of the necrosis organisms are made, healing takes place with remarkable rapidity. Identical results have been obtained in cases of superficial or deep necrosis of the skin after accidents or burns.

It seems that, under the influence of the injection, the organism makes specific antibodies, which act directly against the microbes of necrosis.

The normal serum of horses can be simultaneously employed at the injured places, while the subcutaneous injections of cultures are kept up.

This method, after all, is not new. For several years its application in human medicine has given excellent results.

It is, in reality, a specific action for which are used injections of the same microbes which give rise to the lesions and the antibodies that are formed are also specific.

But, adds Lignieres, there is a point to which I desire to bring special attention; it is not only the specific microbes which can bring useful action in the recovery of wounds, other foreign microbes of different species are also able to produce in the organism antibodies which assist in the recovery. It is, however, better to give the preference to the specific action as it is the surest and most efficacious.

Observations have indeed shown the possible utility of the injections of microbes foreign to the disease. The vaccine for anthrax, injected in a flock, where there was a disease which had nothing to do with the bacteria of Davaine, has for several days arrested the disease, and even exceptionally completely subjugated it. Lignieres has often made the same remarks with the antipasteurellie vaccine. After immunization against bovine piroplasmosis, consisting of intravenous injection of blood from sick animals, in which the parasite had been killed, many times a preventive action against foot-and-mouth disease has been observed. These observations are not coincidences but facts very often repeated.

Another demonstration more evident is shown in the fact that a wound rebellious to cicatrization or having a tendency to become

again infected, will show a hasty march towards recovery by the injection of a microbe, foreign to the sore, the coli bacillus for instance. The reaction that this dead microbe will promote in the organism, will then act very strongly in favor of recovery, by stimulating the normal defenses, by promoting the formation of antibodies and by phagocytosis.

To summarize: the injections of specific microbes, killed by heat or any other method are beneficial to the recovery of the lesions provoked by the same microbes.

The injection of microbes of another species, although less certain, may also have a similar effect.

Upon inquiries made by Prof. Moussu on the subject of doses of the solutions to be injected, Lignieres pointed out that it was not necessary, as in man, to enumerate the microbes. In the case of the microbe of necrosis, he has injected under the skin 100 c.c. of killed culture, repeating 3 or 4 times every other day. No abscess or general reaction took place. After the 5th or 6th day the effect was already visible but if smaller or weaker doses were used, no effect was manifested.



WOUNDS AND SERA. The long series of terrible events accompanying the war, has unfortunately been the occasion for various observations of wounds of all kinds and for their treatment. Aseptic and antiseptic measures, compounds of all kinds have been used, all varying more or less according to the nature of the injury, its pathology, microbiology, etc. In several chronicles as well as in abstracts from other periodicals, I have kept the readers of the JOURNAL informed as much as possible with the current of what was taking place from the booming of the application of tincture of iodine to the more recent serum. A fair idea can be had of the progress and of the changes that have occurred in the treatment of wounds, in human and in veterinary practice, and in several instances deserving attention has been given to the polyvalent serum of Leclainche and Vallée.

This serum which has been the object of high recommendation in human surgery and which, by the published notices in foreign as well as in continental scientific and professional papers, has received at the hands of veterinary practitioners, the welcome it deserves. Our professional contemporaries now begin to record the results obtained from its use.

Among the many publications relating to it, there is in the *Bulletin de la Societe Centrale* a report presented by Principal Veterinary JACOULET, on the treatment of diseased withers, poll evil, cartilaginous quittor, deep wounds of the foot by punctures, castration, abdominal cryptorchism, which show the great benefit that has been obtained by the use of the polyvalent serum above named. It has succeeded in most of the severe wounds under consideration.

From all the cases recorded by M. Jacoulet there is one which appears to me so special that I cannot help referring to it in a concise manner.

It is a case of poll evil and diseased neck. A very severe case, which was treated with the serum only and in which a complete recovery was obtained without any other antiseptics, in twenty-four days.

In a few words the case was this: A seven year old black mare had distemper and as she got over it, showed a swelling, warm and painful in the middle of the shoulder and one at the poll of the head. The first was resolved with a blister, the second became an abscess as big as both fists. It was open-treated and seemed to be doing well when a large and painful enlargement appeared on the anterior half of the superior border of the neck. An incision was made on both sides of the neck. The cervical ligament was extensively diseased. The cervical ligament was excised and a piece thirty-four centimeters was cut off. Necrosed lamellar portions were removed. The occipital bone was diseased and scraped. The wound that remained measured 36 centimeters in length on one side and 16 on the other. It had a depth of 12 centimeters. No antiseptic mixture was used. Only ampoules of polyvalent serum, four in the first days and gradually reduced until the 20th day after the operation. On the 24th day, the wound was entirely closed and the mare at work. She carried her head normally and there was only a slight deviation of the superior border of the neck in the part where the cervical ligament had been amputated.

An ordinary surgical interference in such a case, followed by one of the advocated antiseptic treatments would have required long months to realize the complete recovery that the polyvalent serum accomplished in TWENTY-FOUR DAYS. Aside from destroying the microbes and superficial cellular elements, it differs from antiseptics by giving rise to changes in the wounds which inhibit microbial de-



velopment and gives to the cellular elements of the organism a new strength and vigorous action.

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INTRA-SPINAL INJECTIONS OF GASES. Perhaps the therapeutics of small animals can be benefitted by this new method of treatment inaugurated by Doct. Felix Ramond which is recorded in the *Presse Medicale*.

Experiments made on dogs, two years ago, have shown him that some gases could be introduced with impunity, into the spinal canal, providing their injection did not surpass in volume that of the cerebro-spinal liquid removed first. A greater quantity may give rise to phenomena of temporary compression, which are, however, only mild in character.

The cerebro-spinal system of man tolerates those injections as well as animals. For that reason Doctor Ramond has attempted to treat some painful manifestations, with some rather encouraging results, as for example those of tabes, headache and sciatica, by the intra-spinal injections of sterilized air.

Also by analogy he was tempted to arrest the progress of tuberculous meningitis by bringing the cerebro-spinal nervous centers in immediate contact with sterilized air. The result was relatively fair. It is true that death of the patient took place but there was a prolongation of life that his condition did not justify.

Oxygen is tolerated as well as sterilized air. It was indicated because of its antiseptic qualities in the treatment of cerebro-spinal meningitis which could not be treated by specific treatment, by the anti-meningococcic, anti-parameningococcic and streptococcic sera.

The author records four cases of meningitis from which 40 centimeters of purulent cerebro-spinal fluid were extracted and replaced by the injection of 20 centimeters of oxygen. The treatment was followed by complete recovery, as indicated by the clinical symptoms and the clear appearance of the spinal fluid.

The intra-spinal injection of colloidal gold is supported better than the intravenous; that of oxygen, providing it is not beyond the volume of the purulent fluid removed, does not give rise to special reaction. The combined injection of gold and oxygen seems more efficacious than when only one is used.



ANOTHER TREATMENT FOR TETANUS. It is from the *Presse Medicale* that I extract the analysis of a recent article entitled STUDIES ON TETANUS. After giving in two chapters, general considerations on the development of the infection and on the clinical manifestations of late tetanus, following a preventive injection of serum, the author in a third chapter considers the use of the persulphate of soda in the treatment of the disease.

The destructive action of oxydizing agents and especially of the alkaline sulphates on the tetanic toxin is well known. After many positive experiments made on several hundreds of animals, injections of persulphate of soda were introduced in human medicine and valuable results have been obtained. Out of 33 cases of tetanus which were submitted to the treatment, 16 recoveries were obtained, with thirteen deaths by tetanus and four from other diseases occurring after recovery from tetanic infection. Such results have never been obtained by any other mode of treatment.

The treatment consists of venous injections made once or twice a day, according to the severity of the case, of 20 c.c. of a solution of persulphate of soda, pure and neutral at 5%. These injections can be kept up for 8 or 10 days, regulating them according to the symptoms. They are reduced when the attacks subside or repeated again if they continue.

Immediately after the administration of the injection, the spasmodic manifestations pass away or are extremely reduced. However, some permanent contractions do not seem to be affected by the treatment, the trismus, the contraction of the abdominal muscles and the stiffness of the neck may last for some time.

Because of the repeated failures of the treatment now advocated, perhaps persulphate of soda may be worth trying by veterinarians.

A. LIAUTARD.

The Western New York Veterinary Medical Association held its third annual meeting at Buffalo, N. Y., June 28. A clinic was held in the afternoon after which there was a dinner at the Touraine Hotel. The program in the evening included papers on The Effect of the Milk Law upon the Production of Market Milk; The Buffalo Milk Supply; a discussion of Catarrhal Fever in Cattle; Reports of Cases and discussion of same.

## THE DEVELOPMENT OF IDEAS REGARDING THE PREPARATION AND USE OF ANTI-RINDERPEST SERUM\*

ARCHIBALD R. WARD

Bureau of Animal Industry, Washington, D. C.

While rinderpest is not likely to occupy the attention of the profession as a field problem within the continental United States, yet the disease should afford interest to American veterinarians because of the close parallelism existing between it and hog cholera, especially in connection with prophylaxis by antisera. Hyperimmunization, the use of normal salt solution for peritoneal washing, potency tests, the serum alone method, and the simultaneous use of virus and serum are practices common to the management of both diseases. The literature of rinderpest is widely scattered in publications not readily accessible and consequently not familiar to many in America, and further contains many discrepancies in statement that have only recently been rectified. Consequently a review of the development of ideas concerning anti-rinderpest serum, supplemented by abundant references, is presented for consideration.

The value of immunity in preventing rinderpest has long been known. Gamgee (1) in his book on *The Cattle Plague*, published in 1866, cites the work of Dodson in 1754 in which it was recommended that immunity be induced by inoculating calves with virulent material. This method was widely practiced in Europe and in some cases the death rate was remarkably low. This was particularly true among the cattle on the Russian steppes where the disease ran a mild course.

Koch (2) in 1897 reported to the Secretary of Agriculture of Cape Colony "that blood serum of cattle which have recovered from rinderpest had a certain immunizing effect upon healthy stock when inoculated with it." He drew a large quantity of blood from an immune and injected 100 c.c. of the serum into another animal which received 1.5 c.c. of rinderpest blood the day after the serum injection. The animal remained well for six days when it was injected with 1 c.c. of rinderpest blood to test the immunity and no sickness resulted.

\*Presented at the meeting of the A. V. M. A. Section on Sanitary Science and Police, Oakland, Cal. September, 1915.

Looking back over the span of eighteen years since Koch's work and in the light of subsequent developments, his one experiment with 100 c.c. of serum from an immune animal used simultaneously with virulent blood seems to furnish the fundamental idea of the most practical and economical method of combating rinderpest with serum, even though he disparaged the importance of the result at the time. His subsequent work in South Africa seems to have been concerned with immunization with bile, a method long since abandoned. However, later on Koch visited India in connection with the use of serum.

Turner (3) mentions a method of immunizing animals with defibrinated blood of those that had recovered, devised by Theiler and Pitchford and elaborated later by Danyz, Bordet and Theiler. An animal immunized naturally or by the use of bile was injected with 100 c.c. of virulent blood. After the reaction had ceased, blood was drawn. This when defibrinated, was injected into a susceptible animal. At the same time the nose of the animal was smeared with infective material, and to make infection even more certain it was turned in with sick animals. Usually the animal contracted rinderpest, whereupon it was again injected with 100 c.c. of defibrinated immunizing blood—why, is not clear. In the light of present knowledge it seems superfluous.

Kolle, in cooperation with Turner (3) in 1897 introduced the process of injecting immune animals with successive and increasingly larger doses of virulent blood for the purpose as stated by him of producing a serum of higher potency than that yielded by natural immunes. Successive doses in amounts of 10, 50, 100, 200, 500, 700, 1000, 2000, 3000, and sometimes 4000 c.c. were injected in turn as soon as the reaction from the preceding injection subsided. During the work Turner abandoned the use of defibrinated blood in favor of serum on the ground of the poor keeping quality of blood, which prevented holding for potency tests, and further on account of its greater liability to transmit diseases.

The serum was used for curative purposes on a few experiment animals. It was also used in infected herds where the disease was prevalent and had already caused deaths before serum treatment was given. It appears that the superior potency of the serum over that of a natural immune was assumed from these observations on experiment animals and reports of treatment by "farmers and others" who administered it in the field. There is no mention made

of a test of the potency of serum from the same animal or group of animals before and after the procedure that is now designated as hyperimmunization. Such would be the crucial test of what hyperimmunization accomplishes.

Nicolle and Adil-Bey (4) in 1899 modified the method of hyperimmunization by injecting the immune animal with 4 to 8 liters of blood at one time. They mentioned that it was much more simple to practice immunization and hyperimmunization in one single procedure. Four liters of virulent blood and 25 c.c. of serum were injected. After fifteen days the blood serum of the animal was protective in a dose of 25 c.c. Eight liters could be injected instead of four and the blood was more active, but the activity was not in proportion to the dose of virus inoculated.

The same writers added three per cent of potassium citrate to the blood used for hyperimmunization and found it advantageously replaced the use of defibrinated blood.

Nicolle and Adil-Bey (5) introduced the use of peritoneal washings as a substitute for virulent blood. This procedure was favorably reported upon by Ruediger (6) who showed that it cut in half the item of cost for animals producing virulent blood. Further, he regarded the serum from animals injected with peritoneal washings as more potent than in the case where blood was used.

Kolle and Turner introduced into the literature of anti-rinderpest serum the idea of a prolonged passive immunity conferred by the injection of serum. Turner (3) concludes that 200 c.c. of serum will afford immunity for some two months or more. Kolle and Turner (7) gave five experiments as evidence that large doses of anti-rinderpest serum confer an immunity of many months' duration. Kolle (8) quite recently repeats the statement that 100 to 200 c.c. confers to healthy cattle a condition of immunity continuing for three months. The very limited work from which their conclusions were drawn is not such as to carry conviction and the conclusions have lately been severely criticised (18, 20). Nevertheless the ideas as to passive immunity advanced by these writers have been repeatedly reaffirmed without confirmation and have had a profound influence upon the methods of using anti-rinderpest serum.

The belief that anti-rinderpest serum confers a period of passive immunity was made by Stockman (9) the basis in the Trans-



vaal of a campaign against the disease in which the use of virulent blood was avoided.

Head (10) in writing of the serum alone method observed that the disease would break out again about three weeks after injection. He felt that the expense of reinjecting with serum was a serious matter, and further, under such conditions was not able to determine when the disease was ended on account of its smouldering among animals protected with serum.

Arloing (11) noted that the disease continued to take victims, disappear and reappear, during a period when repeated injections of serum alone were made.

In India the serum alone method as reported by Lingard (14) and by Walker (15) consisted of injecting animals with a protective dose of serum and immediately exposing them to natural infection. Such a use of serum is the same in effect as simultaneous inoculation and gives opportunity for obtaining the maximum possible beneficial effect from the serum. The present writer regards such use of serum as entirely in accordance with known facts.

The serum alone method in the Philippines was used with the idea that it would give a lengthy period of immunity. Jobling (12) places it at two to four months and Ruediger (13) states that the immunity persists two or three months. The writer arrived in the Philippines just at the close of the period when serum alone was used. The idea of the field men engaged in the work was that the serum should prevent attack during a period of several months. As a matter of fact, sickness and deaths even, occurred with disturbing frequency within two weeks after injection with serum. In conjunction with the use of serum, efforts were made to prevent infection by isolation and disinfection. Instances of belief held by certain individuals that the serum protected against attack were common enough. Men would point to the fact that certain animals in a locality where infection was prevalent, did not contract rinderpest after injection of serum was made. However, in these cases it was not possible to show that the animals were not already immune nor that they had actually been exposed to infection.

In this connection facts regarding the transmission of rinderpest are of great interest. The literature of this phase of the rinderpest problem contains scarce and contradictory references to the matter. Ruediger (16) in the Philippines had made the statement that pastures which have been infected by sick animals may remain infected for months or even for years.



The present writer (17) in cooperation with Wood and Boynton attacked the problem and drew the following conclusions:

"1. Rinderpest virus was not shown to have survived beyond twenty-four hours in corrals bare of vegetation but containing water. The conditions under which tests were made included all seasons of the year with accompanying variation in sunlight, rain, and condition of the soil. The amount of shade varied widely.

2. Animals became infected in such corrals within half an hour, twelve hours, and seventeen and one-half, respectively, after removal of the sick.

3. Animals infected with rinderpest were shown to be capable of transmitting the disease to susceptible animals by close contact only during the febrile period of the disease, and most certainly during the period in which the temperature was declining. The disease was not contracted by susceptible animals when exposed to sick animals during the convalescent stage when the temperature was nearly normal.

4. Blood of animals infected with rinderpest was shown in two cases to be infective during the height of the febrile period.

5. The virus in urine, diluted with water and sprinkled on grass, was demonstrated to survive for thirty-six hours, in some instances, but not always, and not for a longer period of time.

6. Feces mixed with water and sprinkled on grass infected an animal twenty-four hours later.

7. Feces and urine diluted with water and kept in a vessel in the shade remained infective for susceptible animals for thirty-six hours, but no longer.

8. No evidence was secured to show that recovered cases transmit the disease.

9. The foregoing facts indicate that the virus of rinderpest perishes soon after being discharged by the infected animal.

10. Nothing in the foregoing experiments indicates that rinderpest virus is harbored for long periods upon the soil of contaminated areas."

The above results drawn from a series of 26 experiments, many of them duplicated, emphatically contradict the idea that rinderpest virus remains alive for long periods outside the body. These conclusions along with clinical observations in the field, point to the danger of the unrecognized mild cases, of which there are many, as a source of infection. Thus an isolated animal injected with serum stands a small chance of coming in contact with infection, except indirectly through the attendant.

Struck with the discrepancy between the claims that serum alone induces a long period of immunity and the hard fact that it did not do so in the field, the writer (18) with Wood undertook

some tests of the matter. At the time no information was at hand to show that anyone had put to test the statements as to passive immunity made by Kolle and Turner and repeated by various authors. The results did not show that hyperimmune serum even in excessive doses, prevented infection. However, in cases where large doses were used, the severity of the attack was so modified that symptoms might not be discerned. Animals injected with serum and exposed to infection immediately contracted the disease nearly within the usual incubation period, and passed through an attack, the severity of which was in inverse proportion to the amount of serum used. The first rise of temperature was delayed, as compared with controls, a fact attributed to the effect of the serum. The results justified a severe criticism of the ideas of Kolle and Turner.

A comparative test was made of hyperimmune serum from animals that were naturally immune before hyperimmunizing and of serum of animals that were susceptible when first immunized. The results indicated that test animals injected with the product from serum herd animals originally susceptible experienced a shorter period of sickness than those with serum from immunes. Ruediger (19) concluded that serum from nonreactors was equal to or very nearly equal to that from animals that had actually contracted rinderpest from the injection of virulent blood. Gibson (21) observes that as a general statement, serum from animals that have recovered from a mild attack is more potent than that from an animal that has recovered from a severe attack. In practice the difference is negligible, for slight deficiency in potency may be compensated for in a larger dose.

While our work was in progress the work of Holmes (20) came to notice. He concluded that a protective dose of serum when injected remained so only for two weeks and even at that time its power was greatly diminished. Thus four out of nine animals died when inoculation of blood was delayed two weeks. Those tested at twenty-one and twenty-eight days all died. Increasing the dose extended the protective period somewhat. He also showed that an active immunity could be conferred to a susceptible animal by the injection of virulent blood together with an amount of serum sufficient to mask all signs of a reaction.

There is no question but that immense amounts of anti-rinder-

pest serum have been wasted by use under conditions where the animals were not immediately exposed to infection. It is believed, however, that most of this serum is now used in the simultaneous method as employed in Egypt and the Philippines or as serum alone under conditions very similar, as in India.

The cost of producing hyperimmune anti-rinderpest serum has even been a hindrance to its use on a scale proportionate to the needs, and further has led to the reduction of dosage to a dangerous point. This has brought the simultaneous method promptly into disrepute, for the first condition of its popularity among stock owners is that their animals shall not die. Turner (3) reported that the serum produced at the Kimberly station sold for \$36 a liter. The cost of production is not given, but presumably in a time of public calamity a government institution would not aim to make an excessive profit. Sample lots of serum from French Indo China and from Japan cost the Government of the Philippine Islands at the rate of \$24 and \$17 a liter respectively. It cost the Transvaal Government \$12.50 a liter, and the Government of the Philippine Islands \$12 a liter to prepare.

Practically the whole of the expense centers around the hyperimmunizing process. However, Gibson (21) seems to be the first writer who questioned the necessity of hyperimmunizing animals before drawing blood for preparing anti-rinderpest serum. He stated that he had abandoned hyperimmunization on the ground that an animal that would tolerate 150 c.c. would tolerate indefinitely large amounts. He regarded the additional reaction obtained by huge doses as just as likely due to the resentment of the organism to the foreign material, as to the virus itself. He felt that it was a great advantage to the veterinary officer in an outlying district to have an inexhaustible supply of immune defibrinated blood, and be quite independent of a serum laboratory.

Shealy (22) also made a similar observation to the effect that just as good results could be obtained with serum prepared from animals after recovery from an attack as when the animals had been hyperimmunized.

The present writer (23) and Wood were impressed by the necessity of having cheaper serum for use by the simultaneous method and undertook field trials of serum from animals that had merely been immunized in the field and not hyperimmunized.

Our previous experience (18) had emphasized the fact of the ease of controlling the severity of the attack by increasing the dose. Thus, we felt that even if immune serum were appreciably less potent we could easily compensate therefor. Further, Wood had noticed as had Gibson and Shealy, that good results could be obtained from the use of nonhyperimmune serum.



FIG. 1. Autoclave with gasoline heating apparatus used in field serum laboratory. Photograph by Bureau of Agriculture, Manila.



The first 141 animals immunized received hyperimmune serum, but subsequently only serum from the animals immunized in the field was used. It was found practicable in the field to use apparatus for collection of serum in such manner that abscesses did not result from its use. To do this in a tropical country under camp conditions and with native assistants I consider to be a notable achievement of the men in direct contact with the work. Figs. 1 to 3 give an idea of how the work was carried on. It seemed preferable to use serum rather than defibrinated blood, for by eliminating the blood cells we thereby reduce the amount of inert material injected. At the end of the immunizing reaction blood to the amount of three liters was drawn from a large percentage of the animals without reference to how strongly they had reacted. Each lot of animals was detained about a month for immunization and blood drawing.

The death rate tabulated for one lot of 429 animals was 1.4 percent; for another group of 1657, was 1.3 per cent. Subsequent to my leaving the Philippines the method was applied to 1900 imported animals with all the disadvantages of their having experienced a trying sea voyage, with a loss of .63 per cent.

In the first few injections we used 200 c.c. on a small lot with a loss of 25 per cent, and 300 c.c. in another lot with a loss of 4 per cent. Consequently we decided upon a dose of 350 c.c. per buffalo (carabao) as they ran, without making nice distinctions as to weight, which probably varied from 600 to 1000 pounds. This would be a dose varying from 35 to 53 c.c. per hundred pounds. We had previously noted that a dose of 48 c.c. of hyperimmune serum per 100 pounds was necessary to protect the most susceptible cattle in the Philippines.

Aside from the small death rate, the immunized animals made prompt recoveries. The result upon the general health of the animals was entirely satisfactory to the owners. Subsequent to the work herein reported, this system of immunization with the voluntary consent of the owners has been extended to other parts of the Islands such as the Province of Pampanga and the importation quarantine station at Ilo Ilo. The funds at the disposal of the Bureau of Agriculture have not been sufficient to meet the demand for the work.



At the outset, the work was conducted cautiously with little consideration of overhead charges. However, when it was in full swing with the time of the men profitably occupied the cost to the Government was 33 cents apiece for 1,056 animals injected in one month. Had hyperimmune serum been employed the cost would have been \$4 per animal for the one item of serum. No expense was incurred for virulent blood, this being obtained from animals at the height of the immunizing reaction.



FIG. 2. Method of drawing blood with vacuum apparatus at field serum laboratory. Photograph by Bureau of Agriculture, Manila.

The effect of an insufficient dose of serum was illustrated by an experience on a large sugar plantation where immunization was undertaken during the existence of an outbreak of rinderpest among hard worked, rundown cattle. One lot of 35 animals received only 100 c.c. of serum due to a misunderstanding of instructions by the operator, and 34 per cent of them died. The dose was raised to 300 c.c. of serum and of one lot of 30 animals 2 died of causes not attributable to the immunization reaction. Another lot of 30 receiving 300 c.c. of serum, lost 4 animals.

While our work was in progress Holmes' (14) publication on the same general subject appeared. He concludes that the serum obtained after natural recovery or after an immunizing reaction is little inferior in potency to that taken after the process of hyperimmunization. Some variations as to effect of hyperimmunizing were noted among the various classes of animals experimented upon. The serum of cattle of the plains of India could not be improved upon by hyperimmunizing with virulent blood of the same class of cattle. The serum of buffaloes could be made twice as potent by hyperimmunizing with virulent blood from buffaloes. This was the greatest improvement found to result from hyperimmunizing. Thus doubling the dose of serum of nonhyperimmune buffaloes would compensate for omitting hyperimmunization.

Holmes further pointed out that virulent blood from an animal being immunized by virus and serum could be employed in hyperimmunizing. Thus an animal may serve to produce virulent blood for hyperimmunization and after recovery from the reaction be bled for serum, after which it may be hyperimmunized, and more blood drawn for serum. The lowering of potency incurred by drawing blood may then be corrected by injecting more virulent blood. The economy effected has permitted doubling the output of the laboratory without any additional expenditure.

Thus the idea of the greatly superior quality of serum from hyperimmune animals that has dominated the thought and practice of workers on anti-rinderpest serum since the work of Kolle and Turner in 1897 must be abandoned. In the field of work of immunizing in the Philippines we reverted to the methods employed in South Africa before hyperimmunization had ever been suggested.

The idea of obtaining a great concentration of immune bodies per unit volume by hyperimmunizing has led workers on rinderpest into using complicated, expensive processes without adequate return therefor. The phrase "impotent serum" seems to have designated that which fell below a certain concentration per unit volume (arbitrarily designated), that which in dose of a given number of cubic centimeters per hundred pounds of animal would not protect against a given amount of virulent blood and which serum therefore was worth less in price than potent serum. Incidentally, the standard of one or two c.c. of virulent blood, against

which varying doses of serum are tested is certainly not the definite unit that is constituted by a given quantity of bacterial culture toxin. It is by no means clear that varying this factor within wide limits, makes any difference. Impotent serum seems to have been that which would have been valuable if the quality had been expressed in terms of so many cubic centimeters as a protective dose per animal. An impotent serum seems to have been that which was used in a dose too small to protect.

The idea of Kolle and Turner in using gradually increasing doses of disease producing blood seems to have been to parallel the practice of injecting toxin, as in immunizing horses against

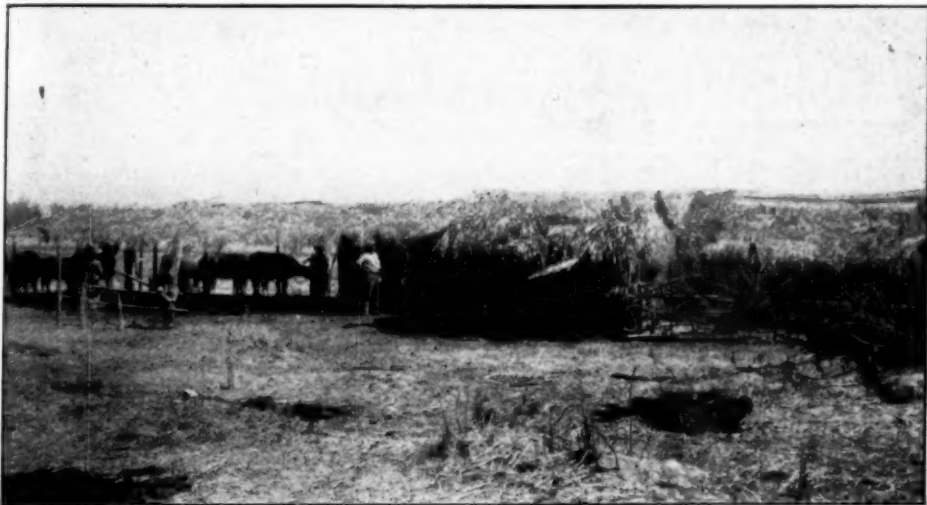


FIG. 3. Field serum laboratory.  
Photograph by Bureau of Agriculture, Manila

tetanus and diphtheria. There is no true parallel between the procedures of making antisera for tetanus and for rinderpest. In the former case the toxin is elaborated in cultures in the laboratory and is available in greater amount than would be elaborated in the tissues of the animal sick of the disease. Also, its introduction into the body may be controlled when a horse is being artificially immunized.

In the preparation of anti-rinderpest serum the phenomena are quite different. It was soon found that a single injection of a large amount of virulent blood gave results in hyperimmunizing

equal to that afforded by gradually increasing doses. Thus, within certain limits at least, there were no serious toxic results in rinderpest as would have followed the injection of a bacterial culture toxin. Later experience has shown that the injection of 1 c.c. of virulent blood will set up an attack during which antibodies are developed to such a degree that the blood in such case is little inferior to that from a hyperimmunized animal.

What really is in virulent blood used for hyperimmunization and what these constituents accomplish has not been worked out. The virus, of which we know little, is there. At the height of an attack, immune bodies whether or not in maximum potency must be present. Thus while injecting virus in virulent blood we are also injecting antibodies. Is there a toxin present in the virulent blood of a sick animal but not comparable in concentration to a culture toxin? Likely, for rinderpest manifests itself in a susceptible animal as a toxemia. No toxin has been prepared and studied in rinderpest in the sense that tetanus and diphtheria toxins are known. Is the toxic element in virulent blood bound to the antibodies?

What constituent in virulent blood is active in producing hyperimmunization in those cases where this process produces a measurable result? Will the virus organisms multiply in the blood of an immune animal, or will they stimulate antibody formation without growth? Is there a relatively weak toxin which stimulates antibody formation? What of the antibodies in the virulent blood? In massive injections they constitute a recognizable factor. At any rate, the results of the injection of virulent blood as a whole, have been over-estimated and therefore none of the factors is of as great importance as formerly supposed.

Whether hyperimmunizing is worth while or not it seems, should depend upon the cost of caring for the animal during the extra time necessary to produce hyperimmune serum and upon the increase in potency accomplished. Religious or other scruples of the natives of India against the drawing of blood in the field may make it necessary to produce serum at a central laboratory; in the Philippines none such exist.

Likewise is discredited the exaggerated idea of the period during which a protective dose of serum alone remains effective. Every day that elapses before exposure to infection results in a dimi-



nution of the protective property that was introduced by the serum. In no case is a susceptible animal, when exposed to infection, protected from invasion of virus and an attack of some degree of severity.

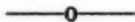
Consideration of ideas held concerning anti-rinderpest serum furnishes subject for reflection in connection with hog cholera and suggests a number of inquiries concerning the use of serum in the latter disease.

What is the potency of hyperimmune anti-hog cholera serum as compared with the non-hyperimmune serum drawn just after immunization? Could immunization in hog cholera be induced by one single injection of the hyperimmunizing dose, with a protective dose of serum as done by Nicolle in rinderpest? If so, what is the minimum amount of virulent blood necessary?

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## LYMPHOSARCOMA OF THE FOWL

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**HISTORICAL:** Only one authentic case of lymphosarcoma of the domestic fowl\* has been recorded in the literature at hand from the library of this institution. This case was found in the heart of a fowl. The heart appeared about normal size. The sectioned surface showed small, roundish, clear areas, which upon microscopic study proved to be a lymphosarcoma.

A sarcoma is a connective tissue tumor whose elements, either because of their number or often because of their size, predominate over the intercellular substance.†

Sarcomata develop either in previously normal tissue belonging to the connective tissue group, as the skin, subcutaneous tissue, intermuscular connective tissue, peritoneum, spinal cord, meninges, or connective tissue of glands.

Sarcomata may also develop in some pre-existing connective tis-

\*Poultry Diseases and Their Treatment, Page 138—Kaupp, *Am. Jr. Vet. Med.*, 1914.

†General Pathology, page 419—Ziegler—William Wood & Co., New York.

sue tumor as a fibroma, myoma, chondroma, and hypertrophic lymphangioma.

The transformation of the parent tissue into tumor tissue takes place through the growth and multiplication of the existing cells. This cell division takes place principally by the process of mitosis, and the mitoses are the more abundant the more rapid the growth of the tumor. In addition to typical mitosis there are frequently observed atypical forms, also nuclear fragmentation or karyorrhexis, and more rarely segmentation.

A sarcoma in its well developed state forms a more or less sharply circumscribed new growth. These tumors may appear in any part of the body where connective tissue is present. There are some tissues in which the sarcoma appears more abundant than in others, these are the skin, fascia, intermuscular connective tissue, bone marrow, liver, intestines and lungs.

The sarcoma when sectioned appears soft to the touch, cuts easily and is easily torn. Microscopically the neoplasm appears more like the developing connective tissue of the embryo or the granulation tissue of inflammation.† The cells of the sarcomas are of various shapes and sizes. They may be fusiform, branched, spheroidal or flat, and at times cuboidal or even cylindrical. They may be very large and possess more than one nucleus or they may be very small and spheroidal in shape and possess but one nucleus. They may somewhat resemble a lymphocyte. The abundance of cells may so cover over the basement substance that it may entirely escape superficial observation. Again it may be so abundant as to give the section the appearance of a fibroma. The cells may be intimately intermingled with fascicules, or, they may be in large open-meshed networks, giving to the tumor the alveolar appearance. The cells always stand in relation to the basement substance, which they sometimes reveal by fibrillar processes continuous with it. These tumors are also richly supplied with blood vessels, and in fact, form an important structural element.

Sarcomas are malignant tumors. They may occur rather early in the life of the animal. As stated before, they are cellular in character, grow very rapidly, are quite vascular and hence succulent and are marked with their tendency to recur when excised. They

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†A text book of Pathology, page 348—Delafield and Prudden—Wm. Wood & Co., New York.

spread both by contiguity of tissue and by metastases. Occasionally a sarcoma takes on a slow growth, under which conditions they are rather dense and, in fact, may remain localized and remain harmless for several years.

The sarcomas by their intimate relations with the blood vessels and also lymphatic structures are prone to spread by metastases especially by way of the blood currents. The richly cellular and vascular sarcomas are prone to hemorrhages, degeneration, and ulceration.

Each tumor is usually made up of just one kind of embryonic cells. In many cases, however, the cell form varies greatly in the growth.

There is a tendency to reproduce in these tumors some of the special characteristics of the tissues in which they originate. Thus, sarcomata of the bone are likely to be osteo-sarcomata; those of the choroid are likely to be pigmented sarcomata; of the brain, gliosarcomata; in fatty tissue, lipo-sarcomata; in cartilage, chondrosarcomata; in gland tissue, adeno-sarcomata; in muscle, myo-sarcomata; in the dura mater, fibro-sarcomata; with lymph structures, lympho-sarcomata; containing giant cells, giant-celled sarcomata; where blood vessels predominate are called angio-sarcomata; with mucoid tissue, myxo-sarcomata; and in muscular structure and containing muscle cells mixed with it, myo-sarcomata; where the cells are held in nests the framework being connective tissue the tumors are called alveolar sarcomata.

In regard to the type of cells, in a simple or in a complex sarcoma, there may be the following: small or large round cells, small or large spindle cells. These four types of cells are the predominating factor in the sarcomata.

The small round cells, of the small round celled sarcoma, are about the size of the small lymphocyte and resemble that type of cell very much. This cell type of sarcomata is usually very malignant. They most frequently occur in the connective tissue of the muscles and fascia, in bone, and in lymph nodes or along the line of lymphatics. In the fowl they most often occur in the intestinal tract and other parts of the abdominal viscera.

The large celled variety possess cells that are very much larger than the last named variety. Their nuclei are large in proportion to the size of the cell and contain prominent nucleoli. They are usually less soft and malignant than the small celled variety.



As stated before there may be retrogressive changes in the sarcomata. These degenerative changes may be fatty degeneration, mucous degeneration, liquefaction, caseation, necrosis, hemorrhage, ulceration and even gangrene.

When the embryonic celled tumor shows a more or less even distribution of the cells without the formation of distinct grouping, it is spoken of as a simple sarcoma.|| Those sarcomata which show a special arrangement and grouping of the individual cells, so that the tumor formation arises which are very similar to the epithelial tumors are of the second class. When secondary changes appear in the cells, in the intercellular substance, and in the blood vessels, giving the tumor a characteristic appearance it is considered a sarcoma of the third class.

The etiology of sarcomata is only partly understood. In mammalian species they occur more often in youth than in old age. As yet too little data are at hand to make a definite statement about birds. It has been found that some sarcomata even commence development in embryonic life, and the origin has been charged up to some local malformation. Very often traumata appear to be an exciting cause. A parasitic origin has been demonstrated. (Ziegler).

In the formation of sarcomata usually one primary tumor is formed and from this multiple tumors result by metastases. However multiple primary sarcomata may occur. This may particularly be the case in the bone-marrow and skin.

Sarcomata are prone to irregular growths and are usually not encapsulated. They grow by contiguity of tissue, by sending irregular extensions out into the adjacent tissue, thus becoming larger without being definitely circumscribed.

As in mammalian species, so in the domestic fowls, the sarcomata may be transplanted. Rous and Murphy experimented with sarcomata from three different sources. The first was a simple spindle celled sarcoma, the second was an osteochondrosarcoma and third a spindle celled sarcoma curiously fissured with blood sinuses and showing a tendency to metastasize to the skeletal muscles. All specimens were originally obtained from the domestic fowl. In their summary they conclude as follows:

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||Ziegler's General Pathology, 1908, Wm. Wood & Co., New York.

"The phenomena of natural and acquired resistance to transplanted chicken tumors strikingly resembles those observed in the case of transplanted mammalian growths; and no more than those do they suggest that the tumors have an extrinsic cause. §

"That there may exist in fowls implanted with chicken tumor a resistance directed against the tumor-causing agent distinct from the resistance manifested against the alien tumor cells has been shown. Both sorts of resistance are present in a fowl in which a tumor has retrograded, the resistance in such an instance being acquired. That directed against the agent is largely specific, giving little if any protection against the agents causing other tumors. There is some evidence that the conditions upon which a fowl's natural resistance depends are the same for the agents causing different chicken tumors."

"It has proved impossible to protect chickens against the agents causing the simple sarcoma by injecting them with dried tumor material in which this agent has been attenuated by heat. The transfer of blood from resistant fowls with growing tumors is in our experience void of effect on the tumors."

One case of lympho-sarcoma has recently been studied in this laboratory which appears to us to be worthy of record. The following is the record:

**CLINICAL HISTORY:** A single comb Rhode Island Red hen two years of age and a member of the farm flock at Iredell Test Farm.

This hen had appeared in excellent condition, except for a slight diarrhea, up to about ten days before she was sent to the laboratory. She had a loss of appetite, diarrhea, and was thin in flesh. The comb and wattles and facial regions were pale and the plumage in a rather unkempt condition.

The hen was placed in the death chamber and killed by the aid of illuminating gas.

**AUTOPSY PROTOCOL:** The bird was of medium size, thin in flesh and other external conditions were as given under clinical history.

Upon opening the abdominal and thoracic cavities the thoracic organs were found in a normal condition. An examination of the abdominal cavity showed almost a total absence of the retroperitoneal fat, which is usually so common especially in hens two years

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§Jr. Experimental Medicine, Vol. XX, No. 4, page 419.

or more of age. The liver was rather under size and a darkish red in color, weighing only 25 grams. The spleen was normal in size and color. There was noted just anterior to the cloaca and involving the whole rectal wall, a tumor measuring 7.5 x 7.5 x 4 cm. Upon opening this tumor it was found that it involved the entire rectal wall giving passage to that viscus through its middle. The sectioned surface of the tumor wall was 1.5 cm. in diameter. There

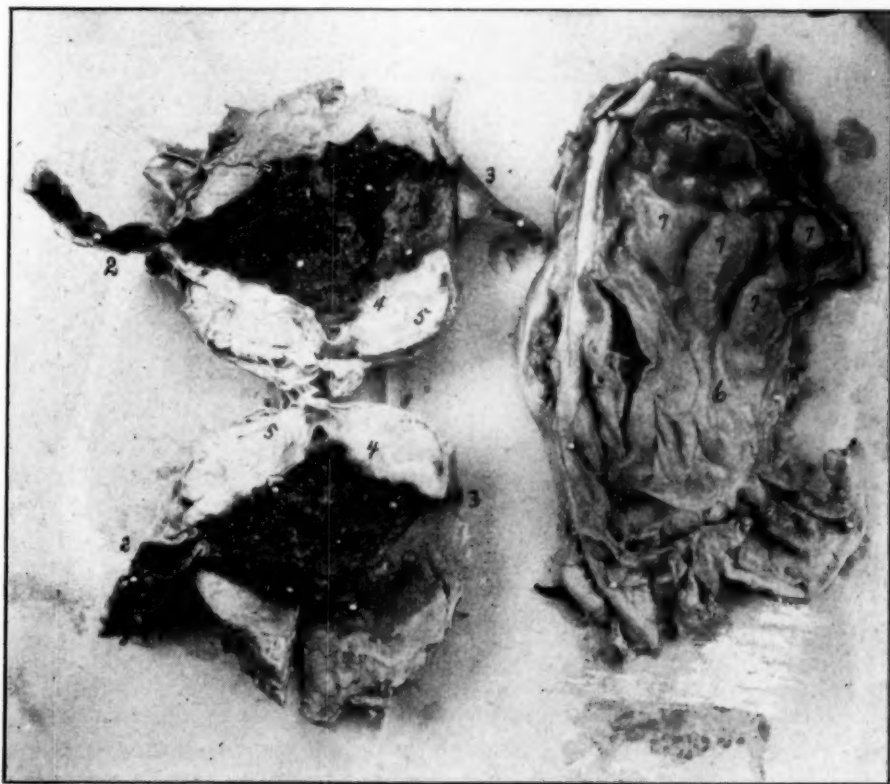


FIG. 1. Lympho-sarcoma. Tumor of rectum and mesentery.

1. Lumen of large intestine—inside of tumor.
2. Uninvolved portion of rectum.
3. A portion of the cloacal mucous membrane.
4. Caseation necrosis.
5. Lympho-sarcomatous elements.
6. The mesentery.
7. Five of the tumors.

was an ulceration of the lumen of the intestine, a mass of necrotic tissue appearing suspended from the wall of the cavity. The cavity was fusiform in shape, measuring 1 x 3 cm. in the widest place.

Anterior to this point and involving the intestinal wall were two other neoplasms which measured  $2 \times 1 \times 1$  cm.

At the anterior point of the right kidney and overlying the adrenal gland was a fourth tumor measuring  $3 \times 3.5 \times 4$  cm. The sectioned surface revealed a solid though soft whitish structure.

In the mesentery of the free portion of the small intestines there were found ten small, rather flat whitish-yellow appearing tumors. They varied somewhat in shape but the majority will fall within the limits of the following measurements which represent the size of two:  $5 \times 8 \times 2$  cm.,  $5 \times 6 \times 1$  cm.

The ovary was in an inactive state. The gizzard, pancreas and proventriculus were normal in appearance.

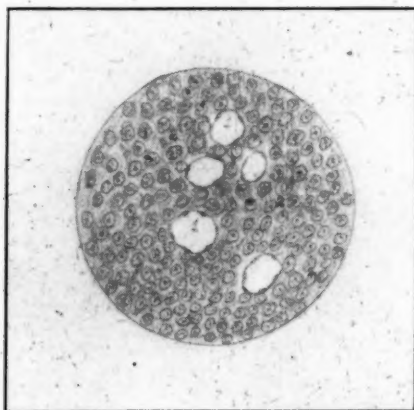


FIG. 2. Lympho-sarcoma of the fowl.  
1. Lymph vessels.  
2. Sarcoma cells.

**MICROSCOPIC EXAMINATION:** The Mesenteric Neoplasms were placed in a ten per cent solution of formaldehyde for 48 hours, then passed through absolute alcohol and then through ether and alcohol and imbedded in celloidin, blocked, sectioned and stained with hematoxylin and eosin and clarified in cedar oil, for study.

The section was packed with small round cells with large nuclei. The cell type appeared to be that of the embryonic variety. Each nucleus contained one or more deeply staining metachromatic granules and many showed distinct nucleoli. These granules were rather small. Here and there was to be noted nuclear fragmentation indicating karyorrhexis. The ground substance appeared to be rather granular and homogeneous and in places rather stri-



ated though no nuclei or cells could be observed except those possessing the large round nuclei of an embryonic type. Numerous blood vessels were observed; these vessels were filled with blood cells and were thin walled. There is no clearly defined limitation of the invasion of the sarcomatous cells into the surrounding tissue. Numerous small vessels, not blood vessels, but containing only an endothelial lining were observed.

A section was made from the **WALL OF THE TUMOR**, involving the large intestine just anterior to the cloaca. On the inner portion there was a layer of necrosing and disintegrating cellular material which may be regarded as caseation necrosis. Under this layer could be recognized a layer densely infiltrated with polymorphonuclear leucocytes mingled with the same small embryonic type cells that were observed in the other tumor sections. The outer layer consisted of densely packed round cells with large nuclei containing one or more small, deeply staining metachromatic granules. The ground substance, in areas where it was observable, was noted to be of a homogenous nature as in the preceding slide. There were a few thin walled blood vessels, most of which were filled with blood cells. There were areas containing numerous small vessels with a single endothelial lining—the lymph vessels.

Sections of the neoplasm from the anterior portion of the kidney and the mesenteric tumors were of the same structure except that these showed no necrosis or polymorphonuclear invasion.

Figure No. 1 shows a photograph of the gross specimen and Figure No. 2 is a drawing through a portion of the more densely packed part. The cells do not show so densely, in order that the outline and details may be shown. There will also be seen some of the typical lymphatic vessels.

The mesentery of the fowl does not contain mesenteric lymph glands as do those of the mammalian class but there are many lymphatic vessels which pass upward from the numerous villi and there is also found lymphoid tissue in the mucous lining of the intestinal tract.

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**CORRECTION.** In the Proceedings published in the May number, page 290, it was erroneously stated that Dr. E. E. Patterson of Detroit, Mich. was a graduate of the Detroit Veterinary College, 1901. It should have been Grand Rapids Veterinary College, 1901.

## NOTES ON THE OCCURENCE OF PETECHIAL HEM- MORRHAGES IN THE LARYNX AND KIDNEYS IN HOG CHOLERA

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For some reason petechial hemorrhages on the mucous membrane of the larynx have received little mention in most accounts of post-mortem lesions found in hog cholera. In the recently published report of the Committee on Diseases, of the American Veterinary Medical Association, on various phases of hog cholera, in the section dealing with post-mortem lesions, no reference whatever was made to these petechial hemorrhages. These appear to form a fairly constant lesion in hog cholera, and do not materially differ from the hemorrhages found in other mucous membranes and in the serous membranes.

We know of no other disease of hogs in which this lesion appears, although hemorrhagic laryngitis is a fairly constant lesion found in hemorrhagic septicemia of cattle. Wherever these hemorrhages are found, it is customary to attribute them to the specific nature of certain diseases. The question arises as to whether these hemorrhagic lesions can be positively attributed to the action of the specific virus of hog cholera or whether we should attribute them to some secondary invaders, which are so common in this disease. In view of the fact that the hemorrhagic lesions found in other organs are practically always mentioned, we can see no reason why the same lesions in the larynx should be overlooked.

The question as to the specificity of the hemorrhages in the kidneys might well be considered in this discussion. In the accounts of the pathogenicity of the old hog cholera organism, *Bacillus cholerae suis*, hemorrhages in the kidneys are said to have been produced by this organism. At least one other organism besides the hog cholera virus and the *Bacillus cholerae suis*, undoubtedly produce lesions of a similar character in certain cases, namely, the *Bacillus necrophorus*.

The writer takes this opportunity to note the fact that petechial hemorrhages have been observed in the kidneys of fetuses found in some pregnant sows that have been inoculated for the pro-

duction of hog cholera virus. This would seem to indicate that if the virus is directly responsible for these hemorrhages, it is small enough to pass through the placental filter. This has been noted in at least two cases, and it is of further interest to relate that in one case no clearly defined hemorrhages could be detected on the kidneys of the sow, but quite a number of them could be seen quite distinctly in the kidneys of the fetus. In the latter case there was no doubt as to the presence of cholera, as most of the other lesions usually found were in evidence, and the sow in question had shown undoubted clinical symptoms of the disease before slaughter.

Petechial hemorrhages in the kidneys undoubtedly occur in a number of pathological conditions in which the kidneys are involved, either directly or indirectly. Post-mortem examinations of hogs, supposed to be immune to cholera, in a number of instances have revealed hemorrhages in the kidneys. Some of these hogs were healthy at time of slaughter, while others had died from some one of several conditions. A number of so-called "hairless" pigs, less than forty-eight hours old, recently examined by the author, showed the kidneys to be almost covered with minute hemorrhages. We may be accused of taking too free license with the term hemorrhage, but microscopical examinations usually reveal such to be the case.

The only references to hemorrhages in the larynx that can be found by a search of current literature include a brief mention by Hutyla and Marek<sup>2</sup> as follows:

"In the pharynx, at the base and on the border of the tongue, sometimes also in other parts of the buccal cavity, and in the larynx, hemorrhagic inflammations of the mucous membrane, with croupous pseudo-membranes and ulcerations, together with a superficial or deep necrosis may very frequently be found."

Also the following from a paper by Dinwoodie<sup>4</sup>, read before the Minnesota State Veterinary Medical Association about two years ago:

"The larynx and trachea in many cases will be covered with a stringy, frothy, adherent, oftentimes blood-tinged and foul smelling mucus. Upon removal the mucous surface is dirty white in color and lustreless. The mucous membranes of the larynx may be thickened and folded, and showing beneath are oftentimes hemorrhages, irregular in outline, number and size. The bronchi may be similarly affected."

In reporting in the same paper upon the lesions found in 350 cases of hog cholera Dinwoodie does not give any data as to the frequency of lesions in the larynx.

These petechial hemorrhages, from a histological standpoint, are possibly the result of some lytic action of the hog cholera virus upon the endothelial cells of the capillaris, or the same effect may result from the combined lytic action of the virus and bacterial



FIG. 1. Larynx of hog showing numerous petechial hemorrhages on the mucous membrane of the arytenoid cartilages. emboli becoming lodged in the finer blood vessels of the tissues where we so frequently find these hemorrhages. Hutyra and Marek<sup>3</sup> make the following mention in this respect:



"According to the histological examinations of Marek, the hemorrhages develop either as a result of bacterial emboli or as a result of a toxic affection of the walls of the blood vessels (the latter especially in the kidneys)."

The hemorrhages in the larynx are usually seen best when a highly virulent strain of virus is being used and the inoculated animals are killed or allowed to die along about the eighth to the twelfth day. The appearance and location of these hemorrhages in different animals does not differ materially, although there is evidence to suggest that they may be transient. This statement is made because of the variations in color, sometimes noted. In some cases the color is a very distinct red, whereas in others, usually in those pigs which have lived a few days longer, this bright red becomes dull, and has a sort of faded or rusty appearance. In some cases only a few hemorrhages will be seen, while in others they will be quite numerous. The majority of the hemorrhages are usually seen in the mucous membrane covering the arytenoid cartilages, but in a few cases hemorrhages are seen on the mucous membrane of the other cartilages, particularly the epiglottis. As a rule the amount of hemorrhage is fairly well divided between the lateral halves of the larynx. The larynx illustrated in Figure 1, was a very pretty specimen and shows a rather extreme case of the hemorrhagic laryngitis herein described.

The lesions found in the larynx in 500 consecutive autopsies show the following:

Hemorrhages	Cases	Per Cent
None	123	24.6
Slight	227	45.4
Moderate	140	28.0
Extreme	10	2.0
	500	100.0

It will be seen that of these 500 cases, hemorrhages in the mucous membranes of the larynx were found in 377 cases, slightly over 75%. These 500 pigs were killed in from five to twenty days after inoculation.

Days following inoculation when killed	Percentage of the 500 pigs killed by days	Percentage of the pigs showing lesions, by days
5-6 inc.	5.4	59.2
7	18.8	67.0
8	21.6	80.4
9	21.8	82.6
10	14.	80.0
11	3.8	78.9
12-20 inc.	4.6	23.3

From the above table it will be seen that the majority of the 500 pigs were killed along about the seventh to tenth days. The table also shows that there is a time (8th, 9th, 10th and 11th days) when these lesions are most frequently found, there being a gradual increase in the percentage of pigs showing lesions up to the 9th day after which there is a gradual decrease. Slight lesions appeared to be more numerous in those pigs killed on the 7th and 8th days. Moderate lesions appeared most frequently in pigs killed on the 9th, 10th and 11th days, whereas no extreme lesions were found excepting in pigs killed on the 9th, 10th and 11th days after inoculation, see the table following:

Days after inoculation when killed	% showing slight lesions	% showing moderate lesions	% showing extreme lesions
7	71.4	28.6	0.0
8	63.8	36.2	0.0
9	54.4	41.1	4.5
10	53.6	44.6	1.8
11	40.0	46.6	13.4
12	50.0	50.0	0.0

A limited number of autopsies have been conducted on hogs dead of cholera following infection in a natural way. Hemorrhages in the laryngeal mucous membrane have been noted in about the same proportion as in the case of artificially infected cases.

**ACKNOWLEDGMENT.** The author desires to give credit to Drs. J. T. E. Dinwoodie and H. C. H. Kernkamp, both of whom have been associated with him during the time that these observations were made, for performing a large number of the autopsies and recording the lesions found.

**CONCLUSIONS.** 1. If we are to continue classifying hog cholera in the group of septicemic diseases, and accept the hemorrhagic

lesions found in the kidneys, lymph-nodes, intestines, bladder, lungs, etc., as a part of the pathological picture of this disease, the hemorrhages found in the mucous membrane of the larynx should also be considered along with the other lesions mentioned.

2. Hemorrhagic laryngitis has been found in about three-fourths of a series of 500 pigs, killed subsequent to inoculation with hog cholera virus, these hemorrhagic lesions being in evidence most regularly in those animals killed and autopsied eight to eleven days after inoculation.

3. In view of the fact that petechial hemorrhages in the kidneys occur in a number of different pathological conditions, veterinarians should be cautious about basing a diagnosis on this lesion alone, even though this lesion does appear in a very large percentage of cases of hog cholera, due to the filterable virus.

1. DIMOCK, W. W. *Journal of the American Veterinary Medical Association*, Vol. XLVIII (New Series Vol. I) No. 2, pp. 213-224.
2. HUTYRA AND MAREK. *Pathology and Therapeutics of the Diseases of Domestic Animals*, Vol. 1. p. 272.
3. *Ibid*, Vol. 1, p. 132.
4. DINWOODIE, J. T. E. Hog Cholera Lesions and Their Significance. *Proc. Minnesota State Veterinary Medical Association* (1914), Vol 1, pp. 37-49.

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## SOME ASPECTS OF THE PHYSIOLOGY OF MILK SECRETION\*

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A brief survey of the theories and scientific investigations upon this subject and an account of the recent experiments upon the increase of secretion of milk due to the injections of animal extracts (Pituitrine).

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INTRODUCTION. Probably no single subject in the whole domain of physiology has attracted more attention, both in ancient and modern times, than the mechanism underlying the secretion of milk by the mammary gland. Since the advent of the hormone doctrine by Bayliss and Starling in recent years many important contributions have been made to our knowledge of this subject particularly with regard to the relationships existing between the mammary gland and the organs of internal secretion. This article

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is a brief, but not exhaustive, summary of earlier experimental work, and a short account of the investigations undertaken with the object of determining the effect on the milk secretion of the injection of substances present in the hypophysis cerebri or pituitary body.

**HISTORICAL. EARLIER VIEWS.** Prior to 1800 A.D., few if any theories of mammary secretions are on record.

M. Richerand<sup>1</sup> 1817 believed that milk was derived from the lymph, while others of his contemporaries believed it to be derived from chyle.

M. Raspail (1833)<sup>2</sup> held the view that the mammary glands have some media of communication with the surface of the stomach "and that they extracted from the alimentary material the salts and organized material which enter into the composition of the milk."

M. Gerod (1839)<sup>3</sup> of Lyons held that "there was an apparatus of vesicles intermediate between the uterus and the mammae, which continue inactive except during gestation and for sometime after delivery when they are excited to activity".

That the composition of the milk was also little understood by these authors is shown by the following quotations: "Casein, the nitrogenized constituent of milk is distinguished from albumen and fibrin by its greater solubility and by not coagulating when heated." This is regarded by Liebig<sup>4</sup> as the chief constituent of mother's blood. To convert casein into blood no foreign substance is required; and in the conversion of a mother's blood into casein no elements of the constituents of blood have been separated out.

M. Raspail (1833)<sup>2</sup> defines milk to be an aqueous fluid holding albumen and oil in solution by means of an alkali or alkaline salt, which he suggests may be acetate of ammonia; and in suspension an immense number of albuminous and oleaginous globules."

**NORMAL STRUCTURE AND DEVELOPMENT OF THE MAMMARY GLAND.** In order that the various theories put forward from time to time to account for the secretion of milk may be better understood it may be well, at this stage, to give a brief description of the structure of the mammary gland.

The mammary glands are composed of a number of lobes which are further divided into lobules. The lobes are composed of convoluted ducts bound together by connective tissue.

These ducts are seen to arise from groups of secretory alveoli.



When traced forward they are found to unite forming the lactiferous ducts, in the human subject about fifteen or twenty in number, which at their points of origin are provided with reservoirs in which the milk collects during lactation. These lactiferous ducts open to the exterior by minute apertures through the teat. In the cow the lactiferous ducts empty into the milk cisterns or galactafor sinuses, which communicate with the exterior through the teat.

The duct walls consist of areolar tissue which contain some non-striated muscle fibers and are lined internally by columnar epithelial cells. These epithelial cells become flattened in the proximity of the nipple.

The secretion of the milk is a feminine characteristic, nevertheless at birth the mammary glands of male and female are very similar and in both sexes there is frequently an enlargement of the glands and an actual secretion of a fluid called "witch milk," during the first few days after birth.

This state of activity disappears after the end of the first or second week. The glands of both sexes remain in this undeveloped state until puberty when those of the female undergo a sudden growth accompanying ovarian functional activity while those of the male remain infantile or atrophy.

The growth of the glands of the female at puberty is largely controlled by the ovaries. If these organs are removed before puberty, the development of the mammary gland, as well as the other secondary sexual characteristics, is repressed.

The hypertrophy at puberty is largely due to an increase in adipose and connective tissue and not to the formation of secretory acini or true glandular tissue.

Unless pregnancy ensues there is no more growth in the mammary glands except a slight hypertrophy which appears at each menstrual cycle. These changes in the human mammae are very slight but in some animals and especially the marsupials, according to J. P. Hill<sup>5</sup>, the amount of growth at each ovulation period is so marked as to render it difficult to distinguish such animals from pregnant ones.

The growth of the mammary glands in the early stages of pregnancy is comparatively slow but it gradually increases in rate and towards the latter part is very rapid. This hypertrophy is due largely to the formation of true glandular tissue and secretory acini.

RECENT THEORIES OF MAMMARY GROWTH. To determine the factors which influence mammary growth during pregnancy has been the object of much research along varied lines. It was formerly supposed that there was a nervous connection between the uterus containing the developing embryo and the mammary gland; that during pregnancy the growth of the mammary gland was stimulated from the uterus, fetus and ovaries reflexly through the central nervous system. This view has been adequately disproven by several authorities.

Goltz<sup>6</sup> and Ewald completely excised the lumbo-sacral part of the spinal cord in a pregnant bitch thus severing all possible nervous connection between the pelvic organs and the mammary glands, which continued to develop as usual, and after parturition the bitch was able to suckle one of the puppies.

Ribbert<sup>7</sup> transplanted the mammary gland of a guinea pig to a position near one of the ears. During a subsequent pregnancy, the gland enlarged and after parturition secreted milk even though it was severed from all possible nervous connection.

Pfister<sup>8</sup> has successfully performed a similar operation upon a rabbit with like results.

Knauer<sup>9</sup> has shown that both ovaries can be removed thus abolishing the phenomenon of "heat". If the ovaries are then transplanted without undergoing complete atrophy, the phenomenon of heat will re-occur even though all possible nervous connection may be destroyed.

Thus, since it has been demonstrated that the pelvic organs do not influence the mammary tissue through the nervous system, we must conclude that the relation between the growth of the mammary gland and the fetus is chemical in nature and that this chemical excitant is carried by the circulating blood.

Hildebrandt<sup>10</sup> has advanced the theory that during pregnancy the developing ovum exerts an "impulse" which acts as a stimulus to growth and at the same time prevents the autolytic disintegration process which he believes produces the specific constituents of the milk.

Lane Claypon<sup>11</sup> and Starling have shown that abortion, or the removal of the fetus, uterus and uterine appendages (Porro's operation), before about the middle of gestation in pregnant rabbits, immediately stops the growth of the mammary glands which quickly

atrophy to their normal size. If Porro's operation was performed after the middle of pregnancy, milk could be expressed from the nipple two or three days after the operation.

Halbane<sup>12</sup> observed from clinical evidence that when the premature death of the fetus occurs without its expulsion there is a swelling of the breasts and soon after, milk secretion. This result is not inevitable, as he has observed that in certain cases milk was not secreted until after the expulsion of the fetus. He has concluded from these observations that the placenta produces the chemical excitant which causes the hypertrophy of the mammary glands and that the appearance of milk in the gland is due to the death or expulsion of the placenta.

These observations as well as those of Lane Claypon and Starling show conclusively that neither the growth of the mammary gland nor milk secretion after parturition can be due to stimulation of the gland at the time of conception since the removal of the fetus during the early part of gestation stops mammary growth and causes the gland to return to its normal size, while the removal of the fetus later in gestation, even before full time, causes milk secretion.

These investigations substantiate Hildebrandt's<sup>10</sup> theory that the chemical excitant of mammary hypertrophy inhibits the secretion of milk and its withdrawal from the system initiates milk secretion.

Miss Lane Claypon and Starling<sup>11</sup> have attempted to determine experimentally which one of the pelvic organs produces this chemical excitant or whether or not it is elaborated by the developing fetus. They have worked on the assumption that the gland or body that produces the chemical "hormone" which stimulates mammary hypertrophy when extracted and injected into virgin animals will cause a similar growth in the mammary gland to that occurring in pregnancy.

They consequently made saline extracts of ovaries, uterus and uterine mucous membrane, placenta and fetus. These extracts were either centrifuged, or mixed with "kiesel-guhr", the expressed juice obtained by means of a Buchner press, and then sterilized by passing through a Berkfeld filter. They were injected either subcutaneously or into the peritoneal cavity of virgin rabbits.

They made several injections during each period of about

fifteen days, trying, as near as possible, to approach the natural condition in which a small amount of hormone is continually circulating in the blood. All of their experiments, excepting those in which the extracts of the bodies or viscera of the fetuses, were used gave negative results.

The experimental rabbits were from eight to twelve months old and in these animals, the glandular tissue could not be detected with the naked eye. In order to compare the rabbits injected with those used as controls the gland tissue was removed and stretched on a cork and hardened, then stained with a dilute solution of hematoxylin and the muscle fibres dissected away leaving only connective tissue and a small amount of glandular tissue surrounding the nipple. In the virgin control rabbits the glandular tissue surrounding the nipple consists of ducts which end blindly and rarely ever occupy more space than one square centimeter. The glandular tissue of some of the rabbits that had received injections of fetal extract covered many times this space and the original ducts had thrown out branches toward the periphery which ended in alveoli. In some cases watery fluid resembling milk could be expressed from the nipples. The glands greatly resembled those of rabbits fourteen to fifteen days pregnant.

They concluded from their experiments that the growth of the mammary glands during pregnancy is due to a particular chemical stimulus elaborated by the fertilized ovum and carried to the mammary glands by the blood stream of the mother; that the amount of this chemical substance produced increases with the growth of the fetus and that it acts to inhibit milk secretion; that its removal by the expulsion of the fetus allows the mammary gland to secrete.

Soon after the publication of their work other physiologists began similar investigations.

C. Foa<sup>13</sup> made subcutaneous injections of bovine fetuses into virgin rabbits for a period of twenty days. After the fifteenth day there was a noticeable enlargement of the mammary glands and on the twentieth day a drop of milk could be expressed from the nipple of the two rear glands. No effect was produced by the extract when it was previously heated to 110°C.

According to the fetal-hormone theory, the injection of fetal extract in lactating animals should inhibit milk secretion. To test this, Foa injected fetal extract into lactating rabbits and found that the secretion of milk was not inhibited.



As a result of his experiments, he concluded that the fetal hormone is not specific to species and is destroyed by heat.

Biedl<sup>14</sup> und Königstein found that the injection of the placental extracts or implantation of placenta into young non-virgin rabbits would cause a histological change in the mammary glands. They also observed that in castrated virgin rabbits, the injection into the peritoneal cavity of an extract of rabbit fetuses, or the intraperitoneal transplantation of rabbit embryo would in all cases produce a more or less rapid growth of the mammary glands. There was an increase in the size of the teat and also an increase of glandular tissue. In the deeper tissues of the gland were found secretory acini, some with wide open lumen and others in an early stage of development. They also observed that the injection of fetal extract had a more powerful effect on the growth of the gland than the intraperitoneal transplantation of rabbits fetuses.

These experiments support the Lane-Clayton Starling fetal hormone theory, but Aschner und Gregoriu<sup>15</sup> are of the opinion that the mammary hormone is contained in both the fetus and placenta. When they injected either the water extract or the ground up pulp of fetuses or placentae into virgin rabbits, they obtained mammary hypertrophy and eventually milk secretion. They observed that the active hormone was destroyed by heat and alcohol. They injected the extract into male animals and obtained mammary hypertrophy and colostrum formation and in very young male animals milk secretion.

The extract of bovine fetuses when injected into female rabbits, produced slight change in the mammary glands. Corpus luteum extract was less effective than the extract of the ovaries. Testicular extract produced some change in the gland and caused the formation of colostrum but did not increase the number of secretory acini nor result in milk secretion. In as much as the mammary hypertrophy could be produced in male animals, they concluded that the ovaries were not essential to mammary growth during pregnancy.

Halbane<sup>16</sup> is of the opinion that the experimental proof of the fetal hormone theory is insufficient and believes that the results obtained after the injection of fetal extract were due to the effect of the period of heat rather than to any hormone in the fetal extract. He is of the opinion that the mammary hormone is produced by the corpus luteum of pregnancy.

Other physiologists ascribe the growth of the mammary glands to the secretory activity of the corpus luteum during pregnancy.

Ancel and Bouin<sup>17</sup> have divided experimental animals into two groups; group I includes animals which have spontaneous periodic ovulation. In this class they have placed man, monkey, cow, horse, swine, and dogs. In group II they have placed animals that have not a spontaneous periodic ovulation but whose ovulation is excited by coitus.

These animals would only have a corpus luteum of pregnancy. In this group we have rabbits, guinea pigs, cats and mice.

When a rabbit is bred to a buck with the vas deferens cut so that pregnancy will not ensue, a corpus luteum will be formed by the graafian follicle ruptured by the stimulus from the coitus. In this manner they were able to form corpora lutea in non-pregnant rabbits.

The corpus luteum thus formed would increase its size, reaching its maximum in about fourteen days and then decline. They observed similar periods of growth in the mammary glands. The first period they called the constructive or kinetic period, lasting about fourteen days, during which mammary growth is very rapid and new secretory acini are formed. This phase they contrast with the constructive phase of the corpus luteum and believe that the mammary growth at this time is due to its secretory activity since they were enabled to obtain it in normal non-pregnant animals in which a corpus luteum had been formed. When the corpus luteum thus formed was destroyed with the cautery, the mammary glands failed to develop.

The second phase they called the phase of secretory activity in which they observed regressive changes in the gland. They believed this phase to be due to the secretory activity of the "myometral gland" which they claimed to have demonstrated exists in the muscular walls of the pregnant uterus.

They described the "myometral gland" as a spindle or polygonal shaped gland composed of different sized cells having a dark granular protoplasm. According to Keifer, this gland reaches its maximum activity about one week before parturition.

Frank and Unger<sup>18</sup> were unable to obtain any change in the mammary glands of white rats after the injection of fetal and placental extracts of the same or of different species. Their experi-

ments upon rabbits in most cases also gave negative results. They were, however, in one case able to obtain a marked change in the mammary gland after the injection of fetal extract. The injections of placental extract for the most part were ineffective, but in one instance, as marked results were found as those obtained from the fetal extract. Out of many experiments they were enabled to obtain positive results in a few instances by the injection of ovarian extracts or the extracts of the hypophysis, (pituitary body).

In one instance they obtained in a normal virgin rabbit a very marked hypertrophy of the mammary gland during the period of heat. At the beginning of this period, the gland was very small as shown by their figure. Twenty-four days later, it was many times as large and corresponded to the glands normally found in animals of the ninth or tenth day of pregnancy. This result is almost as remarkable as those obtained by Starling after the injection of fetal extract. Frank and Unger observed a large corpus luteum in the ovary of this rabbit.

In accordance with the view that mammary growth is stimulated by the secretory activities of the ovaries, we have the work of K. Basch<sup>19</sup>. Into a young female animal Basch transplanted the ovaries of a pregnant dog and obtained, fourteen days later, an increase in size of the mammary gland and in six weeks a marked hypertrophy of the same. Two weeks later, after the injection of placental extract, he obtained milk secretion.

By injecting placental extract into growing virgin rabbits, O. O. Fellner<sup>20</sup> was able to show a marked growth of the mammary gland and a rapid formation of glandular acini. He also obtained, coincident with the mammary hypertrophy, a very marked increase in the size of the uterus and vagina.

From these discordant views of eminent physiologists, it is difficult to draw any positive conclusions. It is probable that the mammary hormone may be isolated from organs or bodies that are not the seat of its manufacture. This in part may account for the discordant results.

That the mammary growth obtained after the injections of fetal extract cannot be due solely to the changes accompanying the oestrous cycle is shown by the experiments of Biedl und Königstein<sup>14</sup> when castrated animals were used and also by Ashner und Gregoriu<sup>15</sup> when male animals were used. Perhaps all the pelvic organs have normally some effect upon mammary growth.

The evidence given for and against the fetal hormone theory thus far has been evidence obtained from normal animals. If we consider the mass of evidence that can be obtained from abnormal cases the problem seems still farther from solution.

Rare instances are on record where men and other male animals have secreted milk which indicates that mammary secretion is not solely a female function.

Virgin girls have been known to secrete milk by allowing infants to suckle their nipples. Other cases are on record where virgin animals have been brought into lactation by mechanical manipulation of the udder.

Several objections to the fetal hormone theory have been advanced by Heape<sup>21</sup>. He quotes a huntsman as stating that virgin bitches were frequently known to produce sufficient milk to permit the rearing of pups; and that the commencement of the secretion of milk in these animals was at the time when they normally would have whelped had they conceived in the preceding oestrous cycle. He uses this argument to show that the development of the mammary gland is due more to the action of the "generative ferment" or "gonodine" secreted by the ovary than to other influences.

A very forcible objection to the fetal hormone theory is supplied by the monotremes. In these oviparous animals, the developing embryo is contained within an egg which does not enter into any connection with the uterine wall. Halbane, however, has pointed out that since the shell is porous there is a possibility that some other substance produced by the developing embryo may diffuse out through the shell and be taken up by the capillary of the uterine mucous membrane and carried to the blood stream of the mother.

Another objection to the fetal hormone theory is given by Lombroso and Balaffio<sup>22</sup>. These authors grafted two female rats so that their circulatory systems were presumably united. These rats became pregnant, but at different times, and gave birth to young, one of them prematurely but the other at full term. Their mammary glands developed independently instead of simultaneously as would have been the case had they both received the same stimulation.

Directly in opposition to this experiment, we have the case of the Bohemian Pygopogus twins Rosa-Josepha<sup>23</sup>, the mammary



glands of both being simultaneously affected by the pregnancy of Rosa who bore a healthy son on April 17th, 1910. The mammary glands of both Rosa and Josepha secreted milk although Josepha had never conceived. They were known to have a common circulation and the glands of Josepha therefore were most probably stimulated through the blood stream.

**THE CAUSE OF MILK SECRETION AFTER PARTURITION.** The actual cause of milk secretion after parturition has not as yet been definitely determined.

Hildebrandt has advanced the theory that the fetal hormone while stimulating the growth of the mammary glands inhibits their secretory activity. He believes that the removal of the hormone from the system by the birth or the expulsion of the fetus allows the secretory activity of the mammary glands to respond resulting in milk secretion. Lane-Claypon, Starling, and others have accepted this theory.

The following are some of the objections which may be offered to this theory. Injection of the blood of the pregnant animal into a lactating animal of the same species does not always inhibit milk secretion. Foa<sup>13</sup> and also Mackenzie<sup>24</sup> found no inhibitory influence resulting from the injection of blood from a pregnant into a lactating animal.

D. Errico<sup>25</sup>, however, obtained a marked inhibition of milk secretion by injection of blood from a pregnant to a lactating bitch. Gaines<sup>66</sup> has obtained similar results.

Placental extract, according to some authors, when injected will cause a growth of the mammary gland, in some instances quite as remarkable as that obtained by the injection of fetal extract. Marked galactagogue properties are ascribed to placenta extract and similar properties have been ascribed to fetal extract. If this be true, we would expect fetal and placental extract to exert a stimulating rather than an inhibitory effect during pregnancy.

Likewise, corpus luteum extract, believed by some authors to be responsible for the growth of the mammary gland during pregnancy has a powerful galactagogue action.

Wing<sup>26</sup> has advanced a theory that while maternity is the prime incentive to milk secretion, yet the immediate stimulus to the production of milk is the "turning of the blood which formally went to nourish the fetus from the arteries of the uterus to the arteries of

the udder." As a result of the increased pressure of the blood in the vessels of the udder the secreting cells are stimulated to greater activity and quickly changed from a dormant to an actively secreting state. Due to the fact that the various processes of secretion are at first incomplete, the first milk secreted differs in composition from that secreted later. It contains less water and more albumen, but a smaller percentage of casein, "as the transformation from albumen to casein is not at this time so complete as later in the period of lactation," this milk is known as colostrum and contains more cells, resulting probably from leucocytes and detached epithelial cells, than normal milk.

McKay and Larsen<sup>27</sup> agree with Wing on this theory and claim that the increased pressure of the blood in the minute capillaries, which form a network in the walls of the alveoli, causes it to swell. This swelling stimulates the epithelial cells to activity resulting in milk secretion.

The saline extract of certain internal secreting glands, and especially of the pituitary body, has powerful galactagogue properties. From these properties one might conclude that these internal secreting glands play an important role in the stimulation of mammary activity. The pituitary body is generally conceded to enlarge during pregnancy so that at parturition we would expect a hypersecretion from this gland. If the fetal hormone theory is correct, we might conclude that the removal of the inhibitory hormone by the expulsion of the fetus would permit the secretions from the pituitary and other secreting glands to stimulate mammary secretion.

This later theory has not been substantiated for no one has proved that the blood of a lactating animal does contain a hormone which is able to stimulate milk secretion and Foa has experimental evidence to show that it does not.

To test this point he used three goats, one virgin and two lactating. They were all etherized and placed side by side on a table, a lactating goat in the center. The carotid artery of the virgin goat was attached by means of a canula to the mammary artery on the left side of the center goat and the mammary vein to the jugular vein of the virgin goat, so that when the clamp was removed the left half of the mammary gland was nourished by the blood of the virgin goat. In a similar manner the right half of the mammary gland was nourished by the blood of the other lactating goat.

The gland continued to secrete milk which was drawn after two and after seven hours from the beginning of the experiment, the milk from the two sides of the udder being kept separate. No differences were observed in the quantity or quality of the milk from the two halves of the udder due to the different sources of blood supply.

Foa has therefore concluded that the blood merely carries nourishment to the gland and does not contain a hormone which stimulates its secretory activity.

According to Gaines<sup>66</sup> the transfusion of blood from a fresh heavily milking goat into a low milking one, fails to accelerate milk secretion.

**THEORIES OF MILK SECRETION.** The nature of mammary secretion is less clearly understood than that of any other of the external secreting glands. Truly, it may be said, that when we attempt to obtain a concise view of the manner in which the various constituents of the milk are secreted we are standing entirely on theoretical ground.

Prior to 1840 the mammary glands were thought to act simply as a passive filter to separate out the constituents of the milk from the blood and lymph. Another theory was that the milk represented decomposition products of the lymph bodies of the blood. The views of Liebig<sup>4</sup>, and Rapsail<sup>2</sup> and others have already been given. It was also believed that the lymph bodies were a source of nourishment to the fetus as well as to furnish the nutriment material for the secretion of milk to nourish the young after birth. "It was supposed that after the birth of the calf, the opening of the uterus from which the food was supplied was closed and then a new opening was formed in the milk gland."<sup>27</sup> These old views have been abandoned for theories more in accordance with ascertained facts.

Neither the blood nor the lymph contains all the constituents of the milk since both lactose and casein are normally absent from blood and lymph. This completely disproves any theory which views the functions of the mammary gland as simply that of a passive filter.

Just how the various constituents of the milk are formed and the source of the material from which they are built up is still not definitely known. While several theories of milk secretion have been advanced, none of them have as yet become well established.

In the actively lactating gland, two distinct phases can be observed. These may be classed as first, the active or secretory, and the second, the resting stage. In the latter condition the alveoli are wide and lined by a single layer of flattened epithelial cells containing but one nucleus. During active secretion, the epithelial cells are long and columnar and some of them project into the lumen of the alveolus; they frequently contain two nuclei and are often charged with fat globules. These fat globules and also many other cells which are probably leucocytes find their way into the alveoli.

In their evolution, the mammary glands were probably developed either from the sebaceous or sweat glands. In the monotremes, they are stated to be modified sweat glands. These animals differ from other species in not having teats and simply exude the milk from scattered pores in the skin. The milk probably passes along hairs which in these regions are arranged in bunches.

In other mammalian orders, the mammary glands are regarded by many (Virchow)<sup>28</sup> as modified sebaceous glands.

Broucha<sup>29</sup>, Bresslau and several authorities, however, regard the mammary glands in all mammalia as modified sweat glands.

There is a tendency for those who hold the view that the mammary glands are modified sebaceous glands to expect mammary secretion to resemble closely the secretion of these glands. For example:—Virchow, who represents the old school of physiologists, believes that the fat in the milk is formed in the same manner as the oil in the sebaceous glands, i. e. from fatty degeneration of the epithelial cells. If this theory were true the epithelial cells would need to be entirely destroyed to furnish the organic constituents of the milk. The colostrum corpuscles were also thought by him to be detached epithelial cells.

There are several sound objections to this theory and the following is one of the most potent:—if the organic constituents of the milk were formed entirely from the epithelial cells an enormous number of these would be necessary to furnish the constituents of the milk.

Heidenhain<sup>30</sup> has shown that all the glandular tissue would need to be renewed at least twice daily and if the epithelial cells alone were used at least five times daily to furnish the solid constituents of the milk. We have no evidence of such rapid cell multiplication as this would necessitate,



The following theory is perhaps the most widely accepted. It was first suggested by Langer and has since been slightly modified and adopted by Heidenhain<sup>30</sup>, Steinhaus<sup>31</sup>, and Broucha<sup>29</sup>, and others. It may be classed midway between the other two theories and is founded on the hypothesis that the secretion of milk is partly due to the disintegration of the cell and partly to the secretory activity of the gland itself.

During active secretion the epithelial cells of the alveoli become columnar in shape and extend into the lumen of the alveolus. According to these authors some of these elongated columnar cells have two nuclei and mitosis is occasionally seen. During secretion the fat globules and one nucleus collect at the free end of the cell which is then snipped off and gives its body substance for the formation of the solid constituents of the milk. The basal portion remains to regenerate the cell. This theory has been most generally known as the cell decapitation theory. To explain the reasons for the cell decapitation in mammary activity the following theory has been advanced:—at the beginning of mammary activity a large amount of blood flows through the arterioles of the epithelial cells furnishing them with a rich supply of food material. This causes them to swell, resulting in the formation of the columnar cells which extend into the lumen of the alveoli. As a result of this swelling the arterioles become compressed, therefore the blood supply is diminished. The supply of blood going to the outer parts of the cell is consequently shut off and they disintegrate and pass into the lumen of the alveoli leaving the basal portion which forms the cubical epithelium. As the products of the secretion are removed, the arterioles are less and less compressed until finally the blood can again circulate freely and supply new nutrient material to the cells which in turn stimulates them to activity. This results in the formation of columnar epithelium which extend into the alveoli and are in turn decapitated to form the next supply of milk.

The same objections may be made to this theory as were advanced against the first mentioned theory, viz. that there is no evidence of such rapid cell modification as would be necessary to replace the outer portions of these cells at each milking. Although only a portion of the cell is supposed to disintegrate to form the constituents of the milk, yet this only differs in degree from the former theory.

Bertkau's<sup>32</sup> objections to the cell decapitation theory are based mainly on his own histological researches. He cut sections of the active tissue of the mammary gland of a cow, dog, and of man and fixed one of each in formalin, and Zenker's fluid respectively. The tissue fixed in Zenker was later sectioned and stained. When microscopically examined, the alveoli were found to contain numerous detached epithelial cells and the phenomenon of "decapitation" was frequently seen. The tissue that was hardened in the formalin presented an entirely different appearance, practically all the epithelial cells were papilliform and extended into the lumen of the alveoli. In some instances, the outer portions of the cells had been torn off leaving the basal portions with a rounded or sometimes a ragged edge. In some sections the outer portions were still loosely attached. The appearance of the cell decapitation was therefore observed much less frequently than in the tissue imbedded in paraffin. Bertkau observed no evidence of mitosis and concluded that the appearance of the decapitation was due solely to imperfect fixation and not glandular activity. Bertkau believes that the mammary glands were evolved from sweat glands and that the secretion of mammary glands is analogous to the secretion of the sweat glands, salivary glands and most other secreting glands and that the solid constituents of the milk are formed entirely as the result of secretory activity of the gland and not from any of the vital constituents of the same. The colostrum corpuscles are believed by those who hold this view to be wandering leucocytes which have passed to the mammary gland from the blood stream.

**CHEMISTRY OF MILK SECRETION.** For many years efforts have been made to determine in what form and manner the various organic constituents are secreted. In spite of all the research that has been done along this line I think I am justified in stating that we do not definitely know how a single constituent of the milk is formed. Several plausible theories, however, have been advanced to account for their formation.

It is possible that the inorganic constituents of the milk are simply diffused from the blood since they occur in all normal blood. That there must be some selective absorption or some arrangement whereby a diffusion of the salts is altered is shown by the fact that they do not occur in the same proportions in the milk as they occur in the blood. Sodium and potassium salts, for example, exist in the reversed proportion to what they do in the blood.

The fats secreted in the milk are at least partially formed from body fat or fat circulating in the blood which nourishes the mammary glands. That this is true to a certain extent is shown by feeding fats of a low or high melting point and thereby lowering or raising the melting point of the butter fat. Butter fat is a special mixture of different neutral fats and differs in composition from any other fat found in the body. This would indicate at least a selective absorption on the part of the cells.

Arguing from the analogy between the mammary glands and the sebaceous glands some authors (Virchow<sup>28</sup>) believe that fat results from a fatty degeneration of the epithelial cells.

During the active secretion of milk, the epithelial cells of the alveoli contain numerous fat globules which collect chiefly in the free end and are stained black with osmic acid. Steinhaus<sup>31</sup>, Nissen, and Heidenhain<sup>30</sup> believe that the decapitation of the epithelial cells permits these fat globules to be set free and thus form the fatty constituents of the milk.

Benda advances the theory that the fat is produced by the secretory activity of the cell and is expelled by a contraction of the cell protoplasm; that the cell itself is not destroyed.

It seems probable that the fat of milk may be formed from either body fat, as is claimed by Soxhlet<sup>23</sup>, Caspari<sup>34</sup>, Henriquez and Hansen<sup>35</sup>, or from carbohydrates as shown by Jordan and Jenter<sup>36</sup>, and also possibly from proteins.

As proof of the latter statement, a dog was fed on pure muscular tissue and according to Ssubotin<sup>37</sup> and Kemmerich<sup>38</sup>, the milk it secreted contained much more fat than could be accounted for in the constituents of the food. It has also been demonstrated that by increasing the fat and protein content of the food, a slight increase in the fat of the milk may be obtained. During starvation in lactating animals the milk yield does not greatly decrease on the first day after the beginning of the fast. Voit<sup>39</sup> cites this as evidence that the milk constituents are formed from epithelial cells which are replenished from the food of the previous day.

Foa's<sup>40</sup> experiments in the perfusion of the mammary gland with normal salt solution containing a known and varied content of proteins, sugars, and fats are very interesting although I do not think at present sufficient work has been done to justify definite conclusions.

He perfused the mammary gland of a lactating goat with a perfusion fluid containing different food constituents.

The gland was retained in situ and kept warm during the experiment and the following are the conclusions arrived at:

The fats of the milk are not formed from carbohydrates or proteins but from fats. Among the fats the mammary gland has the power to choose certain ones and reject others. The casein of the milk is not formed from proteins, differing from those occurring normally in the blood, such as gelatin and egg albumen which is free from globulin; nor from carbohydrates and fats. The production of lactose increases with an increase in the glucose content of the blood indicating that lactose of the milk is formed from the glucose of the blood. The lactose is not derived from the fat or proteins.

According to recent views, the albumen and casein of the milk are formed at the expense of the protein supply of the body or food. Some authors believe casein to be formed from a nucleo-protein resulting from the disintegration of the nucleus of the epithelial cell lining the secreting alveoli. Casein being a phospho-protein may be closely related to the nucleo-proteins.

Other authors believe that casein is formed from albumen by a ferment present in the mammary gland. The comparatively high albumen and low casein content of colostrum as viewed by certain authors is due to the fact that the change from albumen to casein is not so quantitative in the gland immediately after parturition.

Considerable research has been directed toward determining the source of the lactose in the milk and the following are some of the theories and experiments that have attracted attention:

Bert<sup>41</sup> removed the mammary gland of pregnant goats and observed as a result a marked post-partum glycosuria. He concluded that at the time of parturition, the liver was stimulated to produce more than a normal amount of glucose; that when the mammary gland was functioning, this glucose was changed to lactose and secreted in the milk. When the mammary glands are removed, this increase in glucose could not be excreted as lactose of the milk and as a result a hyperglycaemia followed by glucosuria is produced.

Porcher<sup>42</sup> has repeated Bert's experiment and obtained similar results. He also removed the mammary glands of lactating animals



and observed as a result a marked glycosuria. He concluded that Bert's theory was established beyond a doubt. Moore<sup>43</sup> and Parker, and Foa<sup>44</sup> have removed the mammary glands in pregnant goats and failed to obtain post-partum glycosuria. Marshall<sup>45</sup> and Kirkness obtained no post-partum glycosuria in guinea pigs as a result of mammary ablation during pregnancy. The removal of the mammary glands in lactation also failed to cause glycosuria. They concluded that the glycosuria obtained by Porcher could be explained as being normal glycosuria which often accompanies parturition, even without mammary ablation, and the glycosuria obtained after the ablation of the mammary glands in lactating animals as due to the operative shock. In his reply, Porcher maintains that the reason the last mentioned authors did not obtain glycosuria was because they did not test the urine until twenty-four hours after parturition. He maintains that when the increased production of glucose is not used by the mammary gland that its production in the liver is soon checked. He also criticises Marshall and Kirkness for using guinea pigs which he claims are normally pure milkers.

According to his work, in cases of milk fever, either an intense lactosuria or glycosuria or feeble lactosuria follows the stoppage of mammary secretion.

Von Norden<sup>46</sup> found that when glucose was injected per os, in a newly lactating goat, that not glucose but lactose appeared in the urine. Porcher<sup>40</sup> has repeated this experiment using a lactating dog with similar results and cites this as an additional proof that lactose in the milk is formed from the glucose of the blood.

The author has obtained a marked glycosuria but no lactosuria in a lactating goat about four months advanced in lactation by the subcutaneous injection of 4.05 grams of glucose per kilogram of body weight.

Inasmuch as phloridzin diabetes decreases the glucose content of the blood below normal, one might expect it to influence the lactose content of the milk.

Cornevin<sup>47</sup>, as the result of his research has concluded that phloridzin diabetes increased the lactose content of the milk.

Pappenheim<sup>48</sup> and Cremer<sup>49</sup> from similar experiments have concluded that the amount of lactose in the milk was decreased and lactosuria as well as glycosuria was observed. Their results were similar to those obtained by Paton and Cathcart<sup>50</sup>.

According to Porcher<sup>42</sup> phloridzin diabetes does not alter the percentage of lactose in the milk but since the total milk secreted is markedly diminished, the daily yield is decreased in the same proportion as the other milk solids.

The experiments of Kaufman<sup>51</sup> and Magne upon dairy cows would indicate a formation of lactose from dextrose. They analyzed samples of blood taken simultaneously from the mammary vein and the jugular vein during active milk secretion and again when the gland was not secreting milk. They observed from these analyses that the blood coming from the head and also that from the mammary gland, when it was not in a state of active secretion, contained about the same amount of glucose while when the gland was actively secreting, the glucose content of the blood coming from the mammary gland was much lower than that coming from the head.

**EFFECT OF THE INJECTION OF ANIMAL EXTRACTS ON MAMMARY SECRETION.** As has been previously stated, certain organs and glands when extracted and injected exhibit galactagogue properties. The corpus luteum and placenta have been previously mentioned in this regard. To me this field of research has been very interesting as it reveals a possible method used by nature in stimulating mammary secretion. It may lead to some interesting discoveries regarding the nature and manner of mammary secretion. There is also a possibility of eventually obtaining a practicable application of our knowledge derived from this field of research.

Lederer<sup>52</sup> and Pribram were among the pioneers in this field of research. They made a series of experiments upon a goat in which a canula was inserted into the nipple and the amounts of milk secretion measured both under normal conditions and after the injections of extracts under experimental investigation. They found that the extracts of placenta produced a marked increase in milk secretion while ovarian extract was without effect.

In a pamphlet on internal secretion written by Ott and Scott<sup>53</sup> of Philadelphia in 1910, the statement was made that "infundibulin" (20% extract of the posterior lobe of the pituitary) was a powerful galactagogue." In October, 1911 they published the full results of their experiments. As experimental animals they used lactating goats which were bound down and not etherized and the milk secreted by the gland was removed by a water aspirator. They recorded the number of drops secreted during five minutes under

normal conditions and during a similar length of time after the injection of the extract to be studied.

They observed that in one instance the secretion of milk was increased from 5 drops per five minutes to 405 drops per five minutes, by the intravenous injection of five drops of "infundibulin" (a 20% extract of the posterior lobe of the pituitary body). They also tried the action of other animal extracts but found none so powerful as the extract of the pituitary.

The extracts they found that stimulated milk secretion were:—pituitary (the strongest), corpus luteum, pineal gland, and thymus, while they observed that the extract of the ovary, spleen, pancreas, and adrenalin and iodothylin inhibited milk secretion.

They observed that neither the albumen nor the choline constituents of the extracts were responsible for their action.

Upon receiving Ott's notice that infundibulin was a powerful galactagogue Schäfer and Mackenzie<sup>54</sup> began a series of investigations with the so-called galactagogues.

They used mainly lactating cats but lactating dogs were occasionally employed. The milk secreted was led off from the teat either through the nipple or by the exudation method to a drop recorder and a kymographic record taken simultaneously of the milk secretion and blood pressure. In the exudation method the ducts of the gland were incised and a lamp wick was placed in the cut portion. When the milk was drained off directly from the nipple, the teat was incised enough to allow a canula to be tied into it in this way overcoming the action of the sphincter muscle of the teat. The animals were etherized or chloroformed and the anaesthesia maintained by subcutaneous injections of chloral. They used a normal saline extract of the posterior lobe of the pituitary body and arrived at the following conclusions:—

That the extract of the posterior lobe of the pituitary body including the *pars intermedia* and *pars nervosa* when injected intravenously, invariably produced a marked flow of milk within about twenty seconds after the first injection. The flow of milk was rapid at first but gradually became slower and stopped after a period of three or four minutes. A second injection after thirty minutes caused a much smaller secretion than the first and in some cases no response was obtained. They also confirmed Ott and Scott's re-

sults in regard to the galactagogue action of corpus luteum extract.

The researches started by Schäfer and Mackenzie were later continued by Mackenzie<sup>55</sup>.

He found that the action of the extract was not specific to species or even to mammals since the pituitary bodies of cattle, male animals and even birds produced a decided milk secretion when injected into lactating cats. In this regard, it is interesting to note that Herring<sup>56</sup> has demonstrated that the extract of the cod's pituitary body possessed galactagogue properties.

The amount of pituitary extract used by Mackenzie was 5 c.c., which represented the active principle of three ox pituitaries. The extract was found to be insoluble in alcohol and its activity was not impaired by boiling and prolonged treatment with alcohol.

The animal extracts which he found gave positive results were those of the pituitary body, corpus luteum, pineal gland, mammary gland and involving uterus. Of these extracts, the extract of the pituitary body was by far the most powerful.

The injection of pilocarpine and atropine previous to the injection of pituitary extract does not inhibit its action which justifies the conclusion that it acts on the glands directly and not through the nervous system.

Fetal extract had a decided inhibition on the secretory activity of the gland due to pituitrine. This would seem to substantiate the fetal hormone theory.

Schäfer<sup>57</sup> records an experiment performed on a young married lady nursing her second baby, by Dr. W. J. E. Sumter. The baby was entirely breast fed and was beginning to want more milk than the mother was able to supply. From 1 to 1½ c.c. of pituitary extract, representing two grams of posterior lobe of pituitary body, were injected intramuscularly on four different occasions at intervals of two or three days. In all cases except after the first injection, the extract seemed to have an immediate action. The patient could feel the milk coming in with a "tingling sensation", but there was no apparent increase in the yield for the entire twenty-four hours since the baby did not seem to receive more than usual on the days when injections were given.

The following table gives the immediate results of the action of pituitrine:

"Five minutes pumping before injection of 1.5 c.c. of pitui-



tary extract yielded 2.5 drams (9 c.c.) of milk. Five minutes pumping after the injection of 1.5 c.c. of pituitary extract yielded 9 drams (32 c.c.) of milk."

Mackenzie<sup>55</sup> records one experiment with pituitrine extract on a woman, in the tenth month of lactation who was suffering from a mammary abscess in one breast. The healthy breast alone was observed. It was emptied with the suction pump yielding 60 c.c. of milk and immediately an injection of pituitrine was given. The gland was again emptied one-half hour later and 100 c.c. milk were obtained.

None of these experiments give evidence of permanent increase in milk secretion or even a prolonged effect resulting from pituitary injection. In order to determine whether the total amount of milk per day was influenced by injection of pituitary extract, Gavin<sup>58</sup> began a series of experiments upon lactating cows. He divided his experimental animals into three groups. Group A consisted of four cows; two of them were fed 1.8 grams of dessicated pituitary body (about five whole glands) and the other two were fed 3.5 and 5 grams respectively, of mammary gland.

There were also four cows in group B, two of which were injected at 6:45 A. M. immediately after milking, "with seven c.c. pituitrine" (equals 3.5 grams fresh gland or .7 gram of the infundibular lobe). Cows III and IV were injected at 3 P. M. and milked one-half hour later.

In group C intravenous injections of 7.5 c.c. of double strength pituitrine (equal  $1\frac{1}{2}$  infundibular lobe of fresh gland) were given as one injection into cows I and II. Cows III and IV were injected with corpus luteum extract while cows V and VI were used as controls. Gavin has concluded from the results of these experiments that under ordinary farm practice no commercial benefit can be derived from the use of pituitrine since neither the total quantity per day nor the quality of the daily sample of milk was effected by the use of pituitary in any of his experiments.

Some very interesting experiments upon the effect of pituitary extract on the secretory activity of the mammary gland in lactating goats were performed by John Hammond<sup>59</sup>. He used goats in various stages of lactation and the amount of the injection was usually 1 c.c., Parke, Davis & Co. or Burroughs & Wellcome and Co.'s commercial extract of the posterior lobe of the pituitary body, given subcutaneously.

He observed that pituitary extract, when injected into lactating goats has an immediate galactagogue action on the mammary gland but the effect soon passes off; that the fat content of the milk received after injection is increased and there is a corresponding decrease below normal at the next milking. The proportions of the other solid constituents of the milk do not seem to be altered by the injection.

Smaller doses give less milk with a lower percentage of fat than larger doses, but there is a limit in the size of the dose beyond which an increase in amount injected will not result in a corresponding increase in the secretion of milk.

He concluded that the increase in blood pressure resulting from the injection of pituitrine was not responsible for its galactagogue action and that it acted on the epithelial cells of the alveoli and not on the muscles present.

Hill and Simpson<sup>60</sup> observed that by injecting pituitary extract into a lactating angora goat a decided increase in milk secretion could be obtained and that the milk so obtained was abnormally high in fat content. The injection was made either just fifteen minutes before milking or immediately after milking in which case the goats were milked again fifteen minutes after the injection.

In one instance fifty cubic centimeters of milk testing 12.9% of fat was obtained at the evening milking. Immediately after milking the goat was injected with pituitary extract and fifteen minutes later yielded 40 c.c. of milk testing 18% of fat. In this instance 64.5 grams of fat were obtained from the normal milking, and 136.5 grams (72 grams more) were obtained 15 minutes later as a result of pituitary extract injection. There was, however, a corresponding decrease in the milk secreted at the next milking. So that the total secretion per day was not materially increased by the injection. It appears as though a part of the milk which would normally be secreted at the next milking period is obtained as a result of pituitary injection.

Of the solid constituents of the milk only the fat was altered by the injection of pituitrine the other constituents remained constant throughout the experiment.

Hill and Simpson<sup>61</sup> also investigated the action of pituitary extract on milk secretion in the cow and obtained similar results to those obtained when a lactating goat was used.

The injection given the cow was much larger than that previously used by Gavin<sup>58</sup>. The saline extract of eight posterior lobes of the pituitary was given as one injection. In one experiment upon a Jersey cow a yield of 8.3 lbs. of milk testing 7% fat was obtained at the evening milking. The Ringer's solution extract of eight pituitary bodies (post. lobes alone) was immediately afterwards injected into the external jugular vein. Three minutes later the cow was milked again yielding one pound of milk testing 19% fat. About one-third as much fat was secreted as a result of pituitrine injection as was normally secreted before injection. To obtain one pound of 19% fat in milk from a cow is also remarkable. The next morning there was a corresponding diminution in the amount of milk secreted; and also in the percentage of fat it contained.

The effects of pituitrine injections upon the milk secretion in the human subject was likewise investigated by Hill and Simpson<sup>62</sup>, the subject being a young married woman 24 years of age, in perfect health, who was nursing her second child. The procedure adopted was as follows:—

The baby was nursing in the evening, about eight p. m. from both breasts, presumably emptying them. One hour later 1 c.c. of Parke, Davis & Co. Pituitrine was injected into the biceps humeri of the left arm. "Almost immediately afterwards, in about 20 seconds, the patient could feel the milk coming into the breasts, as she described it. The sensation was something between a tickle and a sting and was similar to that experienced when the baby begins to nurse". Ten minutes later the milk was withdrawn from both glands with a breast (suction) pump. Fifty cubic centimeters of milk testing 6% fat were obtained. On three control days when the same procedure was followed except that no injection was given, the following amounts of milk were obtained:—1st, 7 c.c. testing 3.7% fat; 2nd, 20 c.c. testing 2.8% fat; and 3rd, 13 c.c. testing 3.8% fat. On the average of seven control experiments there was .47 grams of fat secreted in the milk from each one of the milkings, while an average of 3.16 grams was obtained from each of the three milkings when an injection of pituitrine was previously given.

There was no way of accurately testing the effect of the injection upon the amount and quality of the milk secreted next morning. According to the mother there was an apparent diminution

in the quantity and the milk acted as a laxative on the body indicating that the quality was also changed.

In what manner pituitrine acts upon the mammary gland to cause the secretion of milk is now not definitely known. It might affect the secretion of milk by alterations in the blood supply to the gland, but this is unlikely since the first injection of pituitrine, which causes a rise in blood pressure, causes also a secretion of milk. A second injection 30 minutes later will cause a fall in blood pressure but will likewise stimulate the secretion of milk.

The two theories of importance remaining are the following:—

1. That the action is on the secretory epithelium stimulating the cells to active milk secretion; 2. That pituitrine acts simply upon the non-striated muscle fibres of the alveoli of the mammary gland causing them to contract and force out the residual milk which they contain; that this milk would not be obtained by regular milking without the injections. According to this theory no milk is secreted as a result of pituitrine injections.

Hammond<sup>59</sup> is an advocate of the secretion theory, while the following experiments of Simpson and Hill<sup>63</sup> would also appear to lend it support.

The udder of a lactating anesthetized dog was incised so that the milk would flow freely from the cut surface if secreted. A moistened lamp wick was inserted into the incised gland and the free end suspended over the side of the animal in order that the drops from the same would fall upon a drop recorder, and be recorded upon a rotating kymograph. A tracing was taken of the blood pressure changes in the carotid artery by means of a mercurial manometer.

An injection of 1 c.c. of a 1% solution of barium chloride (a powerful stimulant of non-striated muscle) was injected into the femoral vein. Immediately afterwards there was a distinct rise in blood pressure and a slowing of the heart but no increase in milk secretion was observed.

After an interval of three or four minutes 1 c.c. of pituitrine was injected which was followed by the characteristic rise in blood pressure and also a distinct increase in the rate of milk secretion.

Since barium chloride does not stimulate milk secretion but does act on non-striated muscle fibres there is good reason for the conclusion that the action of pituitrine is glandular and not muscular.



Schäfer<sup>64</sup> is of the opinion that the proof of the glandular (secretory) theory is insufficient and from histological evidence favors the muscular hypothesis.

Gaines<sup>65</sup> has concluded as a result of his researches that "pituitrine has a muscular action on the active mammary gland causing a constriction of the milk ducts and alveoli with a consequent expression of milk." That the flow of milk produced by pituitrine is dependent upon the amount of milk present in the gland and that there is no evidence of any true secretory action.

He also believes that the capacity of the udder is greater than the volume of milk drawn at one time and that practically all the milk is present in the gland as such at the beginning of milking.

No one has yet demonstrated the presence of non-striated muscle in the mammary gland capable of contraction. Pituitrine may act on both the muscles and the glandular epithelium of the mammary gland but my researches upon the lactating goat indicate that it has a glandular action.

By carefully summing up this article it can be seen that there is a very rich field for research and many important discoveries yet to be made in the physiology of mammary secretion.

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# A NEW HOST FOR *FASCIOLA MAGNA*, BASSI. TOGETHER WITH OBSERVATIONS ON THE DISTRIBUTION OF *FASCIOLA HEPATICA*, L. IN CANADA

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Pathologist, Veterinary Research Laboratory, Agassiz, B. C., Canada.

In December, 1915, the writer received a deer's liver, which was kindly sent to him by Mr. Kermodé, Director of the Provincial Museum. On examining this liver eighteen large flukes were discovered. It was thought that they must be *F. Magna*, and two of them were forwarded to Dr. Ransom of Washington who confirmed the determination.

The interesting part of this discovery lies in the fact that the coast deer is *Odocoileus columbianus*, and therefore that it is a new host for *F. magna*. Dr. Ransom informs me that there are no records published or otherwise in the Bureau of Animal Industry, of its occurrence in *O. columbianus*. The identity of the deer was established beyond a doubt by Mr. Kermodé.

According to Hall, 1912, "The occurrence of the large American fluke on the Pacific coast is a matter of speculation at present." Stiles and Hassall, 1908 give the following hosts for *F. magna*: *Bos taurus*, *Ovis aries*, *Equus caballus*, *Boselaphus tragocamelus*, *Cervus canadensis*, *C. dama*, *C. elephas*, *C. virginianus*, *C. unicolor*. The accompanying photograph of the section of the deer's liver shows the great amount of damage done to that organ.

On cutting into the liver the ducts were found to be dilated into pockets, which in some cases were quite large, and in comparing their diameter with the enlargements caused by *F. hepatica* a striking difference will be noted. Both flukes cause a blackening and granular appearance of the bile within the ducts, but in the case of *F. magna*, the pockets which sometimes contain three or four flukes naturally hold more of the "inky" bile.

As to the mortality caused in deer by *F. magna*, it is hard to estimate, however, the injury to this particular liver is quite apparent. Texada Island in the Straits of Georgia is the place the liver came from. A visit to this island was undertaken in January with Dr. White. Unfortunately the expedition was unsuccessful owing to bad weather, only one deer was shot, and no flukes were

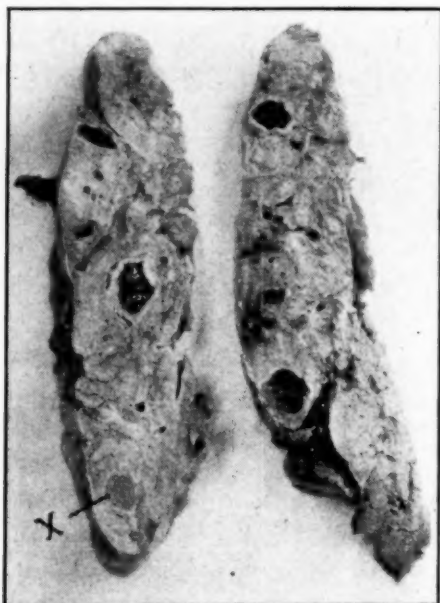


FIG 1. Liver of *O. Columbianus*. Note enlarged ducts, and fluke cut across at point X.



FIG. 2. Sheep's liver. Note flukes at point X.



found. According to the inhabitants of this island a considerable percentage of the deer are parasitized by flukes, and at this time of year (January) show a marked unthriftiness. It would be interesting to compare the clinical picture in deer with that of sheep. As the intermediate host for *F. magna* is unknown, this island seems to me to be an ideal place to investigate the matter, and further work is contemplated.

In all the literature concerning *F. magna* I have come across, no mention is made of the mortality it causes among deer. Ward, quoted by Hall, says: "In importance it stands hardly second to



FIG. 3. *Fasciola magna*, Bassi.



FIG. 4. *Fasciola hepatica*, Linn.

*F. hepatica*". The statement probably refers to domestic animals. Hall says: "Generally speaking this fluke does not seem to make the impression on the health of cattle that *F. hepatica* does on the health of sheep." "It seems to be rather rare in sheep—" Though *F. magna* is no doubt a dangerous parasite, it seems to me that with regard to farm animals it cannot be as easy for them to acquire as *F. hepatica*, otherwise our records would contain more references to it, especially in eastern America, where it has been known for many years.

MEASUREMENTS OF *F. magna*. Fourteen specimens were measured, the average length was 45.4 mm. and the width, 18.5 mm. The eggs measured  $140\mu$  in length and  $100\mu$  in width. These last measurements correspond exactly with those given by Railliet.

*Fasciola hepatica*. This parasite has taken its toll among the coast sheep in British Columbia ever since the early settlements began.

In some districts flockmasters have suffered considerable losses. These losses do not come anywhere near those of European countries where the percentage is much heavier. Neumann gives a mortality of 60 to 75% in some years; not in single flocks but for whole provinces. In British Columbia there are some tracts of low ground where the keeping of sheep has had to be abandoned. In other localities where it is hard to find a flock free from flukes, sheep have been kept year in and year out. Dr. Bruce, Inspector in charge of meat inspection for Vancouver tells me that practically all the sheep coming from one of the river deltas are flukey, yet sheep have been kept there for many years at a profit. It would appear from the above, that land may be lightly or heavily infested in a more or less permanent way. However, the character of the seasons undoubtedly influences the degree of infestation. It may be of interest for me to cite a case which came under my notice. Three years ago I went to see a small flock of sheep, they were badly infested and a number of animals had died. The land on which the sheep grazed was on the bank of a river. Some drainage improvements were suggested, and the owner was especially cautioned against letting his sheep graze along the river front during low water. Since that time the losses in the flocks were light, but in December, 1915 the trouble returned. The sheep at this time only numbered twenty-three, three had died. The symptoms presented were quite marked, dropsy under the jaws, puffy eyelids, dropsy of the abdomen, anaemia and emaciation in a few together with a listless expression. The owner was recommended to slaughter his flock. This was done on December 20th, and on post-mortem every liver had to be condemned, also four of the carcasses. All the animals were more or less hydraemic, but the meat set well. Fig. 5 shows a yearling with a puffy face and eyelids, on the right of the photograph an old ewe. When this animal was killed the abdomen was found to contain fully two gallons of fluid. The young animals were found more affected than the old which seems

characteristic of distomatosis. The most fatal time for the disease in British Columbia is from late December to the early part of March.

Dr. F. Torrance, Veterinary Director General, informs me that he is unaware of *F. hepatica* being found in any other part of Canada. According to Hall the same thing is true for the northern States bordering on the Atlantic; there is only one reported finding on Long Island.



FIG. 5.

Hosts for *F. hepatica* in British Columbia. In sheep kept on the low lands bordering on the Pacific. In cattle, two records, one by Dr. Bruce, one by myself. In deer, *O columbianus* recorded by Mr. Kermode.

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The Wisconsin Veterinary Medical Association will hold its next meeting at Menominie, Wis., July 26 and 27.

The Washington State Veterinary Medical Association held a meeting at Mt. Vernon, Washington, June 21 and 22.

## RECIPROCITY\*

J. W. SALLADE, Auburn, Pa.

It would seem that if reciprocity could be brought about in a practical form it would cure a multitude of embarrassing things that lead to controversy in this association. If there could be devised a method by which our standards of examination and other requirements—subjects covering eligibility to our classes,—a common system of rating, uniform registration laws, equal penalties for violations, etc., covering the practice of veterinary medicine and surgery in all the states of the union; then, and only then, would it be possible to realize reciprocity with equal mutual rights or benefits, to be yielded or enjoyed between individuals so engaged throughout this wide territory, the domain of the United States.

As is well known, the regulation of the practice of veterinary medicine and surgery is a state function and every state that has undertaken the regulation of this branch of endeavor, has its own legislation creating its Board of Veterinary Medical Examiners and defining their duties, and in many instances enumerating the branches or subjects to which applicants shall submit in a theoretical and practical examination. Besides other conditions that create conflicting circumstances all of which would have to be brought into one harmonious connected whole, a uniform system of regulation.

To do all this would necessitate the repeal of all state laws now on the statute books and the re-enactment of uniform laws, a most difficult and hazardous undertaking. It must be remembered that most if not all of such legislation is secured under difficulties and in many instances is crude and unsatisfactory, yet appreciated for the recognition it affords the profession.

Frequent attempts are made in every session of the legislature to so amend these laws as to afford some constituent, otherwise barred from the fold, an opportunity to get his name on the register. The interests of such applicants are approved by legislators for political reasons and our laws are thus a subject of constant attack.

To attempt a radical change, in order to obtain uniformity is fraught with grave danger of losing rather than gaining prestige. If the attempt is ever made the line of attack must be made aggres-

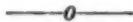
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\*Presented at the meeting of the Association of the Veterinary Faculties and State Examining Boards of North America, September, 1915, Oakland, Cal.



sively all along the front, in every state of the Union, in the same bill. An attack so comprehensive that it will embrace every contingency, to meet the demands, not only of the colleges but of the public and politicians as well.

State Examining Boards are limited in scope by the acts creating them. In view of this fact it would be a relief could all such bodies be made operative under a uniform law. The danger, however, of tampering with such proposed legislation renders the effort of doubtful feasibility.



### SHOULD THE STATE BOARD EXAMINATIONS INCLUDE RECENT DEVELOPMENTS IN VETERINARY SCIENCE?\*

A. D. KNOWLES, Missoula, Montana.

The object of State Boards of Veterinary Examiners is first, to protect the public from being imposed upon by unqualified and incompetent persons holding themselves out as veterinarians and, secondly, to maintain a standard of professional qualifications and moral respect which will give the veterinary profession a proper place and respectable standing in society.

Veterinary science is progressive and those who ally themselves with that profession must be progressively studious if they remain entitled to recognition and service therein. He who assumes upon the receipt of his diploma that he has finished his studies, that he has acquired the necessities for his lifetime in professional knowledge, will fall far short of his possibilities as a veterinarian, and his usefulness and standing among mankind is limited.

Because of the neglect of his professional qualifications, the veterinarian who follows his vocation as a business rather than as a profession must come, sooner or later, to be measured by the public as a professional degenerate and one not entitled to high and honorable standing among men.

He who has not kept strictly in touch with the developments of the science of veterinary medicine during the last decade or two

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\*Presented at the meeting of the Association of the Veterinary Faculties and State Examining Boards of North America, September, 1915, Oakland, Cal.

is less qualified, and his opinion and service along veterinary lines deserves less recognition than that of the well read and intelligent farmer or the recent graduate of an agricultural college.

The veterinarian who is not familiar with recent developments in veterinary science is not acquainted with sero-diagnosis and sero-therapeutics by which many contagious and infectious diseases are now controlled and even eradicated and by which many benign diseases are successfully treated. He is not acquainted with well known vaccines which insure prophylaxis against several infectious deadly animal plagues. He is not acquainted with numerous well known microorganisms which insidiously cause diseases and animal losses which are amenable to treatment or prevention by a correct knowledge of their existence. He is not familiar with many vegetable, alkaloidal, ptomain, and leukomain poisons that are known to the science of today. He is not familiar with the well known principles of asepsis, antiseptics and disinfectants as they exist today. In fact the veterinarian who has not been a student during the past ten years has no correct knowledge of the wonderful developments which have been produced by the pathological, chemical, biological, bio-chemic, and botanical laboratories, and he is surely not entitled to the professional dignity and respect that belongs to the veterinary profession of the present time.

Following this discussion, it is needless for me to state that the answer to the title of this paper should be in the affirmative. There can be no argument in the negative unless it should be considered that veterinarians who graduated ten or more years ago have not had the opportunities to acquire the knowledge of the *Recent Developments of Veterinary Science*. This consideration, however, deserves no attention when we consider the advantage of keeping abreast of the times through our splendid veterinary journals, the many splendid bulletins published by the various departments of the United States Department of Agriculture, by the various state experiment stations, by the various State Veterinary Medical Associations, and by our own A. V. M. A. with its fund of up-to-date reports as published in the annual proceedings. There are also the ever new and increasing text books full of recent development of veterinary science, and the newer and better systems of learning about the science previous to the newer developments.

My experience on the State Board of Veterinary Examiners has been that veterinarians who have been in practice for several

years, for the most part, pass better examinations than the recent graduate, or at least the graduate just from college without having had the advantage of actual practice; and this seems to me to be perfectly natural. This must be true, because the practitioner should lose nothing of what he has gained during his college career and, if he is doing justice to himself, he will have been adding to that knowledge every day and year he is in practice.

In my opinion, the state board that does not include recent developments in veterinary science in its examinations does not do justice to the state which it serves, and it certainly does a gross injustice to the public which looks to it for protection against incompetent veterinarians.

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DIPHTHERIA FROM CATS. An outbreak of diphtheria in an orphanage has been traced to cats. Sixty-nine cases, which were the great majority of those which occurred, came from the boys' house. After many investigations and precautions, such factors in the production of the disease as sanitary defects, contaminated water supply and food were eliminated. Realizing that there must be some carrier of the bacillus, the physician in charge decided to turn his attention to the cats, and took swabs from all of their throats. On bacteriologic examination it was found that the four cats from the boys' side of the orphanage were suffering from diphtheria; the four cats from the girls' side, although showing other microorganisms in profusion, did not prove to have the true diphtheria bacillus. The cats were destroyed, and after that only ten more cases of diphtheria occurred. They occurred within a few days, showing the patients had already been infected when the cats were destroyed. There have been no more cases since.—*London Letter, Jour. A. M. A.*

The seventh annual meeting of the Central New York Veterinary Medical Association was held at Syracuse, June 29. In the forenoon a clinic was held at the infirmary of Dr. Turner. In the afternoon papers were presented by Drs. Stevens, Tuxill, J. A. Pendergast, and Danforth.

## CLINICAL AND CASE REPORTS

### ROUND-CELLED SARCOMA IN THE HEART OF A DOG\*

W. REID BLAIR, D.V.Sc., New York, N. Y.

During the latter part of October, 1914, I was asked to examine an aged Boston Bull terrier, owned by Dr. B., a surgeon connected with the Post Graduate Hospital. I found a very fat asthmatic dog suffering from bronchitis, with chronic cough, and with a very hard wiry pulse, and distinct heart murmurs, induced by leaky heart valves.

The animal was at this time about twelve years old, very active, and until a few weeks before had rarely been ill. As the asthmatic symptoms and chronic cough were doubtless due to the cardiac condition, I prescribed tr. strophanthus three times daily in doses of four drops. This drug produced a very laxative effect on the bowels, and as the bowel condition did not improve after a few days this drug was discontinued and tr. digitalis substituted, in three drop doses. After four days of this treatment improvement was noted in the asthmatic symptoms, and the cough became less frequent and distressing. As the animal's food had for many years been made up of bread, milk, rice and vegetables, I changed the diet to one of meat, with the result that while the dog lost some fat, he showed great improvement in health and spirits. The treatment of the heart was continued at frequent intervals for fourteen months, but although I did not see the dog from October, 1914, till January 1st, 1916, I had, however, been kept informed as to his condition.

The owner asked me to come again January 1, 1916 and I now found the dog not greatly changed in physical condition, perhaps slightly thinner, but with distressed breathing, and an almost constant racking cough, and considerable congestion of the larynx. I suggested codein 1/6 grain every five hours, also pertussin was used. These drugs had no appreciable effect on the cough. The animal's appetite was good, and he was always eager for his food.

The heart pulsations were hard and irregularly intermittent, the lungs congested. We could detect a large area of dullness at the upper and anterior part of the lungs on the left side. I suspected

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\*Presented at the meeting of the Veterinary Medical Association of New York City, March, 1916.



a case of tuberculosis, and after detecting a nodule on the border of the liver, this opinion was strengthened. However, I was not able to positively convince myself, since in the absence of anemia, emaciation, weakness, ascites, and intermittent fever, tuberculosis, in my experience, has been a rare condition. I then considered sarcoma of the lungs and finally convinced Dr. and Mrs. B. that the dog was suffering from either tuberculosis or sarcoma, and that there was no hope of relief, after which they reluctantly consented to have the dog destroyed.

This was done on January 7th, 1916 and the post-mortem examination was made immediately afterward, the owner, Dr. B., being present.

The following gross conditions were present:—

Upon opening the thoracic cavity a tumor mass of globular form was found firmly adherent to the superior auricular borders of the heart. This tumor, about  $4\frac{1}{2}$  inches in diameter, was also attached quite firmly to the trachea and esophagus, and surrounded the pulmonary veins. The heart was greatly hypertrophied, the ventricles were dilated, and the auriculo-ventricular valves showed extensive vegetation and imperfect closure.

The lungs were congested, and showed the production of fibrous tissue as a result of chronic bronchitis, and an excessive amount of pigmentation was present throughout.

The liver was enlarged and congested. A nodule, 2 inches by  $1\frac{1}{2}$  inches, of dark brown color and of firm consistency, was found on the posterior border of the left lateral lobe.

The kidneys showed chronic congestion, and the capsules adherent in several places. Other organs showed no gross changes. Sections of the tumor mass of the heart were taken for pathological histology examination by Dr. B., and later the microscopic examination of the section showed the tumor to be a *small round-celled sarcoma*, the most malignant of the sarcomata. The section of the liver nodule showed this to be an hemorrhagic infarction, with considerable fibrous connective tissue.

In this connection, it may be of interest to consider some common characteristics of these new growths described as sarcomata.

A sarcoma is a more or less malignant tumor consisting of cells, similar to those of embryonal connective tissues. From the beginning of the growth of a sarcoma, a certain type of round or spindle-

cell structure persists, the cells not transforming to more highly developed tissue.

The type of sarcoma varies greatly; certain forms are made up of round cells; others of spindle cells; others of spindle cells and fibres.

The sarcomata are composed essentially of cells, between which a small amount of intercellular substance, usually of a fibrillar form, is present. As a rule, this intercellular tissue is comparatively insignificant, and is seen with difficulty. Sarcoma grows chiefly by interstitial expansion, though a certain amount of peripheral infiltration is characteristic of the more malignant forms. Though sometimes appearing to be encapsulated, the tumor is not always sharply circumscribed from the surrounding tissues.

Sarcomata are usually poorly supplied with blood vessels. The circulation of the tumor is peculiar and irregular. Not only are the blood vessels few, but in many cases those present are imperfect in structure, consisting of an endothelial coat with a few supporting connective-tissue fibres.

Considerable parts of the tumor may be without blood vessels, but contain sinuses or clefts through which the blood circulates in intimate contact with the tumor cells.

Sarcomata may appear in any tissue or organ of the body. A few observers have claimed to find in and among the cells of the tumor certain parasitic protozoa, but nothing definite is known of the nature of these bodies. Many cases appear to follow traumatic injury, but the majority arise independently of any recognizable cause. Sarcomata usually occur as rounded, more or less lobulated tumors. Upon section they are usually pinkish grey in color. The sarcomata vary through all degrees of malignancy. Some are rapidly fatal; others liable to recur after excision. Soft, moist tumors are more apt to be malignant than firmer and more fibrous ones. The general tendency of all the sarcomata is to recur, and the majority produce secondary growths by metastasis. The round cell type most frequently recur through metastasis. Metastasis takes place almost exclusively through the blood channels, and is most rapid when the blood circulates in indefinite spaces in the tumor mass and is free to detach and transport the cells to other parts of the body.

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## AN UNUSUAL MILK FEVER CASE

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M. H. REYNOLDS, University Farm, St. Paul, Minn.

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A very valuable imported Guernsey calf, calved about three months previous, was being fed for a milk record. She showed symptoms suggestive of milk fever on the morning of June 14th. When seen by the writer at about 8:30 o'clock a. m., her temperature was 99.8; pulse about 60, and weak; respirations very shallow. No feces were passed during the night. Milk flow had been greatly and suddenly reduced.

This cow rapidly developed typical symptoms of milk fever. She was lying in the usual position with a tendency to hold the head with a peculiar curve in the neck or around the flank in the usual position. Skin sensation was entirely lost and there was only a rather slow response at the eyes which had a peculiar glassy stare.

On trial with plain water it was found that the cow swallowed easily and a dose of linseed oil, turpentine, and aromatic ammonia, was given about 9 a. m. The udder was inflated about 9:30. The cow made a steady improvement following the air treatment. A mild catharsis developed in the latter part of the afternoon. On the morning of June 15th the cow was quite comfortable and in good condition. Uneventful recovery followed.

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## BOTRYOMYCOSIS\*

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R. W. GANNETT, Brooklyn, N. Y.

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Botryomycosis is defined by Hutyra and Marek as a chronic infectious disease of horses which may, in exceptional instances, occur in cattle. It usually results in the formation of local tumors resembling fibromata and occasionally in the formation of metastasis in the internal organs. It is caused by the botryomyces fungus.

The infectious new growth caused by this fungus is known as a botryomycoma or mycofibroma. It is a neoplasm that occurs very frequently in the horse. It is often malignant in character. Its most common occurrence is in the form of the schirrous cord in the gelding and the cold or deep seated abscess of the levator humeri muscle at the point of the shoulder of the draft horse. It occurs less

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\*Presented at the February meeting of the Veterinary Medical Association of New York City.

commonly in the pectoral region, at the elbow, skin or subcutem in any part of the body, udder, nasal mucous membrane and rarely in bones. Like actinomycosis it is a wound infection disease but metastasis though it often occurs seems to be less common than in the so called lumpy jaw of cattle.

Botryomycomata have been observed in swine and recently in man.

**BACTERIOLOGY.** Formerly the botryomyces fungus and the staphylococcus pyogenes aureus were thought to be identical, but more recent investigations seem to disprove this idea.

The staphylococcus according to investigators quoted by Hutyra and Marek and also by Fröhner forms goldish yellow cultures at any temperature. The botryomyces fungus grows only at a temperature of 18° Centigrade. They are alike in form, take the same stain and have the same grape-like arrangement in clusters. Their development on gelatin is not the same and blood from rabbits immunized with staphylococcus failed to agglutinate the botryomyces fungus. The botryomyces organism possesses pyogenic properties but it also causes chronic productive inflammation of connective tissue experimentally, a property which is not possessed by the staphylococcus.

**BOTRYOMYCOMA OF THE SPERMATIC CORD.** Most cases of schirous cord may be regarded as due to the botryomyces fungus. Infection no doubt takes place at the time of castration. A hard tumor appears sometimes at once but often months or even years after the testicles have been removed. Adhesion takes place between the stump of the spermatic cord and the tunica vaginalis. Growth is gradual. There may or may not be a purulent discharge. Sometimes the growth assumes a malignant character and involves the sheath skin of the scrotum, thigh or abdomen. The cut surface shows numerous purulent or muco-purulent areas and also the smaller yellowish sand like foci of infection.

**BOYTRYOMYCOMA AT THE SHOULDER.** Botryomycoma involving the levator humeri at the point of the shoulder is commonly called cold abscess or shoulder abscess. Its appearance is often sudden and may in many cases be attributed in part to contusion from the collar. After the initial acute inflammation has subsided, growth is gradual but continuous. If neglected the enlargement may become enormous and incurable by involving the carotid artery and jugular vein. It may even extend to the periosteum of the scapula and



humerus. A botryomycoma in this region seldom if ever fluctuates. It rarely or never recovers permanently without surgical interference. It is apt to recur if strenuous measures are not taken to destroy the organism which is its cause.

European writers have recorded cases of generalized botryomycosis affecting such organs as the lungs, kidneys, diaphragm, spleen and liver. Most of them were horses which had schirrous cord.

**TREATMENT OF SCHIRROUS CORD.** Treatment is entirely surgical. The patient should be secured in a recumbent position and anesthetized. The entire schirrous cord should be removed by dissecting well into healthy tissue. The emasculator or ecraseur may be used to divide the spermatic cord. If any of the neighboring skin or subcutaneous connective tissue is involved this too must be removed. Hemorrhage is sometimes troublesome.

In operating upon cold abscess at the shoulder local anesthesia is sufficient. A single bold vertical incision is made down to the central pus cavity. Often there are two or more pus cavities which may or may not communicate. *It is never safe to rely upon ordinary antiseptics for disinfection; either slough out the diseased fibrous tissue with an escharotic or sear thoroughly with the actual cautery.* Botryomycomata in other parts of the body should be operated upon by total extirpation or vertical incision methods, according as they resemble the type represented by the schirrous cord or the cold abscess at the shoulder.

**CASE REPORTS.** A gray draft mare was presented with an enormous botryomycoma involving the entire pectoral region. The growth had become so large that the collar could not touch the point of either shoulder. There were several discharging seton wounds as well as scars from both line and point firing. The growth was completely removed in three separate operations with the aid of stocks and local anaesthesia. Hemorrhage was severe. The structure of the tumor resembled that of the schirrous cord. Cicatrization required six weeks. The mare has been working two years and there is no sign of recurrence of the condition.

A five year old draft gelding in poor condition was operated upon for schirrous cord. He had been passed as sound by me six months previously and at that time showed no enlargement at the scrotum. Shortly after the operation two round fibrous tumor like growths appeared just above the hock of the affected side. They were incised, contained pus, were cauterized and healed promptly.

Soon fibrous nodules of various sizes appeared at the shoulder, all contained pus but healed under treatment. The animal carried a slight temperature at times. He was mallein tested repeatedly but never gave a reaction. About six months after the schirrous cord operation he was in very poor condition, limbs stocked, staring coat, appetite poor and unable to do steady work. Rest, arsenic, nuxvomica and poly-bacterins did not improve his condition. While under treatment a fluctuating abscess of the groin of the affected side appeared. It was lanced, contained foul smelling pus and the wound healed properly but for several months the animal remained in very poor condition, worked half time and had the appearance of a worn out, debilitated old horse. He is now (two years after the operation) in good health and good general condition.

A roan draft gelding was operated upon for a large deep seated cold abscess back of the right shoulder blade. The condition had been noticed for several months. A single vertical incision about eight inches long was made. Ordinary antiseptics were used and the wound healed in about six weeks. The animal was sent home, went to work and in ten days an enormous cold abscess appeared on the opposite side at the point of the shoulder. The owner became discouraged and disposed of the horse.

A bay horse was presented with a large botryomycotic growth involving the skin and subcutem of the left shoulder, withers and side of the neck and of the chest back of the left elbow. There were several discharging fistulous tracts. He was considered incurable and was not treated.

A black gelding showed two round botryomycomata of the skin of the shoulder as large as English walnuts. They were incised and contained muco-purulent material. The cavity was disinfected and packed with cotton saturated in tincture of iodine. Healing took place but soon more growths appeared. I lanced at least twenty small botryomycotic abscesses in two months and eventually was obliged to remove a section of diseased skin and sub-cutem five inches square. The wound was cauterized with the red hot iron and normal healing took place. A tumor as large as a hen's egg soon appeared on the opposite side about half way from the point of the shoulder, to the withers. This was incised, contained pus, the wound was cauterized and healed normally. The horse finally went to work, but the entire treatment had taken between three and four months.

A bay ten year old gelding was operated upon for a cold abscess of the left shoulder of two months' duration. A single vertical incision was made into the pus cavity. No escharotic or actual cautery was used but the wound was treated with ordinary antiseptics. The wound continued to discharge slightly for some time and never healed properly though the animal was put to work.

Four months later I was called to see a large botryomycotic tumor on the right branch of the lower jaw of this horse. There were two small ulcers discharging a non-smelling pus. On examination the third lower molar of the affected side was found to be loose. The animal was placed upon the operating table and anesthetized. The tumor was cut away. Its structure resembled that of the schirrous cord in that there were numerous areas of purulent liquefaction and also the small gritty foci of infection. Several pieces of loose bone were removed. The tooth was punched out but was odorless and showed no visible lesion. The old fistula at the point of the shoulder was opened. The botryomycotic fibrous tissue was thoroughly cauterized with the actual cautery. Convalescence was uneventful and there has been no recurrence of botryomycomata after eighteen months at work.

These case reports have been selected with the view of illustrating metastasis and malignancy in botryomycosis and particularly to emphasize the fact that drainage and ordinary antiseptics are not to be relied upon but must be supplanted by the actual cautery, powerful escharotics or total extirpation of the infectious new growth.

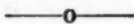
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THE PATHOLOGY OF INFECTIOUS DISEASES OF ANIMALS—MOORE.  
REGIONAL VETERINARY SURGERY—MOLLER-DOLLAR.  
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The joint meeting of the California State Veterinary Medical Association and its Southern Auxiliary was held at Los Angeles, Cal., June 21 and 22. Those participating in the program were Doctors Archibald, Iverson, Meyer, Thompson, Wood, Hurt, Donnelly, Boucher, Irons and Beach.

## ABSTRACTS FROM RECENT LITERATURE

URETHRAL OBSTRUCTION IN A FOAL. C. W. Cartwright, M.R.C. V.S. *Veterinary Record*.—A five weeks old valuable Shire foal was making unsuccessful efforts to urinate. On examination an obstruction was indistinctly felt in the urethra above the scrotum. With a large sized dog catheter, attempts were made to dislodge it. Several trials were useless, only material of a cheesy consistency was left at the end of the instrument. Objections were made by the owner to an operation which was suggested. The use of the catheter was again resorted to and after a while, by persevering, the instrument was felt to pass the obstruction and about half a pint of urine escaped. The flow stopped as soon as the instrument was withdrawn. With directions of careful catheterism being renewed, the foal was left for the night. The next morning he was seen in great pain, which was rapidly increasing. The bladder was evidently distended to the bursting point and immediate relief was necessary. Urethrotomy was performed at once. A gush of urine escaped; the foal made great effort and suddenly the flow ceased, the bladder had ruptured. The little fellow died shortly afterwards. At the post mortem, there was found a calculus of cheesy consistency, about the size and shape of a pigeon's egg, which was firmly lodged in the urethra. The bladder was empty with a large laceration, the mucosa being covered with thick mucous deposits. There was also slight nephritis. All the other organs were healthy. A. LIAUTARD.



THE USE OF INTRAVENOUS INJECTIONS OF CAMPHOR. E. Fischer. *Recueil de Médecine Vétérinaire*, Vol. 92, p. 99, 1916. For affections of the respiratory tract, contagious or otherwise; intravenous injection of a solution prepared as follows, is recommended. Dissolve 8 grams of sodium chloride in 1 liter of water and sterilize by boiling. Add drop by drop, a saturated alcoholic solution of camphor, until a white flocculent precipitate appears. Filter. The dose for a horse varies from 0.9 to 1.5 liters according to the size of the animal. The injection should take 5 or 6 minutes, and should be repeated in grave cases after 12 or 24 hours. There is a violent and short reaction (excitation). The entire treatment consists of 2 to 6 injections, according to the gravity of the disease.

This seems to be a medication having the properties of neosalvarsan—but infinitely less severe. It is being experimented with in several other infectious diseases. BERG.



**SUPPURATIVE ARTHRITIS OF THE HOCK—RECOVERY WITH BIER'S METHOD.** Roy Student and Veter. Dupuy. *Presse Veter.* The horse "Souris" had received a punctured wound on the internal face of the left hock. He was very lame and in three or four days had all the symptoms of suppurative arthritis of the injured hock. The local and general symptoms were characteristic and alarming and as the case was at the front of the battle, with frightful atmospheric conditions of rain, mud, snow, ice, etc., recovery seemed almost impossible and the horse was condemned to be destroyed. Before doing it, however, Bier's method was attempted. A rubber band, such as used in human surgery, for hemostasis and a fine cotton roller, slightly extensive were used and applied one on the top of the other above the hock of the injured leg, the rubber band first as it was rather short and the cotton over it to complete the action of the other. This apparatus was to be left on six hours. The careful description of the case which showed a gradual improvement brought the writer to the conclusion that after thirteen days of treatment, this suppurative arthritis of the hock, rarely amenable to favorable results, was cured by the application of Bier's method. LIAUTARD.

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**EPITHELIOSIS INFECTIOSA AVIUM, CONTAGIOUS EPITHELIOMA, CHICKEN POX, DIPHThERIA, ROUP, CANKER.** O. V. Brumley and J. H. Snook. *The Veterinary Alumni Quarterly, Ohio State University, Vol III (1916), p. 81.*—A large number of birds were presented at the clinic of the Ohio State University for treatment. An autogenic bacterin was prepared from cultures made from the lesions and used as a curative agent. This proved satisfactory while other methods of treatment proved very unsatisfactory. As a result of this method of treatment a series of experiments were undertaken to determine the etiology of the disease and the value of the bacterin as a method of control. As a result of these experiments which are reported in detail, the authors arrived at the following conclusions:

"1. From the bacteriological experiments we have made and the good results obtained from vaccination, we are of the opinion that we have been dealing with one disease only. Owing to the confusion in names which have been used to designate this disease, we have concluded to give it a new name—Infectious epitheliosis of birds (*Epitheliosis infectiosa avium*.) This indicates involvement

of the epithelium both of the skin and mucous membranes. It was thought that inasmuch as we find so many variable forms of the disease that this name would be more appropriate than any combination of names so far suggested.

"2. Our work extending over a period of six years convinces us that typical infectious epitheliosis is due to a combination of two factors: (a) filterable virus; (b) secondary invading organisms which vary in kind but of which the so-called 'Bacillus diphtherae columbarum' of Loeffler, appears to be the most important.

"3. The filterable virus is the necessary primary invader which lowers the birds' resistance and thus prepares the tissues for invasion by the secondary organisms. Neither factor alone will cause the typical disease.

"4. The excellent results derived from the use of a vaccine made from the secondary organisms, both in prevention and treatment, are due to controlling the secondary infections which cause the serious complications. If these are controlled, infection due to the primary virus is mild and soon disappears. (There is a remote possibility that the filterable virus is contained in the vaccine. We have no evidence that this is or is not the case. The presence of the virus in the vaccine would indicate its growth with the other organisms on the cultures. This would be contrary to our present knowledge of filterable viruses. This point will be investigated.)

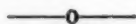
"5. The therapeutic dose, as indicated by the large number of birds treated, is 1 c.c. for the average adult bird. Younger and smaller birds receive a lesser amount.

"6. The immunizing dose found most satisfactory is 1 c.c.

"7. No bad results have followed when larger doses have been administered.

"8. Reports received to date indicate that vaccination is equally efficacious in the treatment of infectious epitheliosis in turkeys."

C. P. FITCH.



SULPHUR POISONING. H. D. Jones, M.R.C.V.S. *Veterinary Record*.—Seven pounds of sulphur were found missing from a bag. It had been given indiscriminately to some 13 horses to cure mange. When seen by the writer eleven had abdominal pains. Two had already died. The pains were not acute; there was a paddling of the legs, a constant looking around at both flanks. The temperature varied: 104° in some, 101°, 102° in others. The mucous membranes

were pale. Two animals died while the writer was there and the post-mortem revealed a strong smell of sulphuretted hydrogen. There was severe gastro-enteritis; the heart and lungs were ecchymotic. A large amount of sulphur was found in the cecum. In two stomachs the amount contained was estimated to be 8 ounces in one and 30 in the other. The treatment was to have all the animals rugged up with blankets and bandaged, friction to the skin, stimulants, whiskey, strychnine, linseed gruel, etc. None of the animals refused food until shortly before death.

Results: Thirteen animals were affected and seven died.

LIAUTARD.

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A CONTRIBUTION TO THE STUDIES OF RABIES IN CATTLE. Borelini, Alberto (Brisighella), Contributo allo studio della rabbia nei bovini, (II Moderno Zooiatro, 1914, p. 141). Abstracted by Ascoli (Mailand) *Zeit für Immunitätsforschung*, No. 12, March 1, 1916, p. 567.—Borelini cites several cases of rabies in cattle in which the entire course of the disease was marked by the absence of violent symptoms, and by the presence of paralysis of the hind quarters. The infection was apparently taken up by the animals from a well in which a rabid dog had fallen but a short time before. The diagnosis in one case was confirmed by microscopic examination, and in other cases by animal inoculation.

REICHEL.

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THE PRESERVATION OF LIVING RED BLOOD CELLS IN VITRO. Peyton Rous, M.D. and J. R. Turner, M.D. (From the Laboratories of The Rockefeller Institute for Med. Research). *Jour. of Exp. Med.*, Vol. XXIII, No. 2, Feb. 1, 1916, p. 219.—SUMMARY. The erythrocytes of some species are much damaged when handled in salt solutions, as in washing with the centrifuge after the ordinary method. The injury is mechanical in character. It may express itself in hemolysis only, after the cells have been kept for some days. It is greatest in the case of dog corpuscles, and well marked with sheep and rabbit cells. The fragility of the red cells, as indicated by washing or shaking them in salt solution is different, not only for different species, but for different individuals. It varies independently of the resistance to hypotonic solutions.

The protection of fragile erythrocytes during washing is essential if they are to be preserved *in vitro* for any considerable time. The addition of a little gelatin ( $\frac{1}{8}$  per cent) to the wash fluid suf-

fices for this purpose, and by its use the period of survival in salt solutions of washed rabbit, sheep and dog cells is greatly prolonged. Plasma, like gelatin, has marked protective properties.

Though gelatin acts as a protective for red cells it is not preservative of them in the real sense. Cells do not last longer when it is added to the fluids in which they are kept. Locke's solution, though better probably than Ringer's solution, or a sodium chloride solution, as a medium in which to keep red cells, is ultimately harmful. The addition of innocuous colloids does not improve it. But the sugars, especially dextrose and saccharose, have a remarkable power to prevent its injurious action, and they possess, in addition, preservative qualities. Cells washed in gelatin—Locke's and placed in a mixture of Locke's solution with an isotonic, watery solution of a sugar remain intact for a long time,—nearly 2 months in the case of sheep cells. The kept cells go easily into suspension free of clumps, they pass readily through paper filters, take up and give off oxygen, and when used for the Wassermann reaction behave exactly as do fresh cells of the same individual. The best preservation solutions are approximately isotonic with the blood serum. If the cells are to be much handled gelatin should be present, for the sugars do not protect against mechanical injury.

Different preservative mixtures are required for the cells of different species. Dog cells last longest in fluids containing dextrin as well as sugar. The mixture best for red cells is not necessarily best for leukocytes.

A simple and practical method of keeping rabbit and human erythrocytes is in citrated whole blood to which sugar solution is added. In citrated blood, as such, human red cells tend to break down rather rapidly, no matter what the proportion of citrate. Hemolysis is well marked after a little more than a week, but in a mixture of 3 parts of human, 2 parts of isotonic citrate solution (3.8 per cent sodium citrate in water), and 5 parts of isotonic dextrose solution (5.4 per cent dextrose in water), the cells remain intact for about 4 weeks. Rabbit red cells can be kept for more than 3 weeks in citrated blood; and the addition of sugar lengthens the preservation only a little. The results differ strikingly with the amount of citrate employed. Hemolysis occurs relatively early when the smallest quantity is used that will prevent clotting. The optimum mixture has 3 parts of rabbit blood to 2 of isotonic citrate solution.

M. J. HARKINS.



**TWIST WITH UNUSUAL SYMPTOMS.** A.V.O. in France. *Veterinary Record*.—This mare had been ridden fifteen miles and brought home without having shown anything unusual. After being in the stable five minutes, she was seen crouching as if preferring to lie down and was slightly tympanitic. She was taken out and walked around. She did not seem to be in much pain but the tympanitis became so severe that her breathing was rendered very difficult. The pulse was hard and quick. Temperature 101°F. She lay down, but did not attempt to roll. While preparations were made to tap her, the tympanitis subsided and no interference was necessary. There was no improvement otherwise, the pulse became weaker, it could hardly be felt, the mucous membranes were injected. Occasionally the mare gulped but there was no attempt to vomit. In the evening, she dropped dead after an illness of five hours. The usual treatment for colic had no effect. The postmortem revealed a twist in about the middle portion of the small intestine. Except at the actual twist there was very little inflammation. There was also a rupture of the mesentery through which the twist had passed. The entire absence of any symptoms of pain, the extreme tympany and the continual regurgitation made the record of the case interesting.

LIAUTARD.

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**CARCINOMA OF THE ADRENAL GLAND IN THE HORSE, WITH HEMORRHAGE INTO THE ABDOMINAL CAVITY.** E. Fröhner. *Monatshefte für praktische Tierheilkunde*, Vol. 27, p. 102, 1916.—As is well known, diseases, especially tumors of the adrenals occur not infrequently in man (Addison's Disease, Bronze Disease). According to the recent investigations of Fölger, Horne and others, pathological anatomical changes in the adrenals in domesticated animals are just as frequent. This is especially true of tumors of the adrenals. Fölger (on the pathology of the adrenals in domesticated animals; this *Monatshefte*, Vol. 20, p. 165, 1909) found 46 adrenal tumors in 300 horses, and 9 in 200 dogs. I have met no cases of adrenal diseases in animals; for this reason the following case of adrenal carcinoma, leading to internal hemorrhage into the abdominal cavity is worthy of notice.

An extremely unsound and decrepit horse was suffering with impaction colic and was treated accordingly. Five hours after arrival there was a sudden general depression, extreme heart weakness, staggering, and death with symptoms of internal hemorrhage.

Autopsy showed as the cause of death, a tumor twice the size of a man's head, next to the left kidney, with a 3 cm. tear in its anterior end out of which blood clots poured. In the abdominal cavity there were 12 liters of fluid blood, in the omental cavity there were six fresh blood clots each as large as a fist. On cutting the tumor, it was seen to consist of grayish yellow tissue, alternated with blood clots. Part of the tumor wall was formed by the cortical layer of the left adrenal. Microscopical examination showed the presence of a carcinoma.

BERG.

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LIVER FLUKE DISEASE, ITS TREATMENT AND CONTROL. J. Marek. *Berl. Tierarztl. Wochenschr.*, 1916, v. 32 (7) 17. Feb., pp. 73-77; (8), 24. Feb., pp. 85-90; (9), 2. März, pp. 97-101—Marek has made a number of tests of substances of a bactericidal and parasiticial nature, which are believed to be taken up by the intestine and returned in the bile, the following substances being tested: quinine hydrochloride, arsenic, chloroform, trypanblau, benzoic acid, helminthol, urotropin, menthol and thymol. All the above were administered in appropriate doses to steers or to sheep infested with liver fluke, and in all cases the flukes were found to survive the treatment.

Better results were obtained with male fern preparations. Filmaron (aspidinofilizin, an active principle of the ethereal extract of male fern), intravenously, into liver, etc., even when repeated for a total of 400 c.c., was found ineffective for steers in 100 c.c. doses of 1 per cent solution, but 50 c.c. doses of the 5 per cent solution intravenously one day, followed by 75 c.c. the next day, and 100 c.c. each day for the next three days appears to be rather effective against *Fasciola hepatica*, killing the larger number and leaving only a few individuals. Neither this preparation nor anything else was effective against *Dicrocoelium*, the small lancet fluke. French and Hungarian extract of male fern and a German male fern preparation called Fasciolin were tested on a series of experiment cattle and sheep to the number of 47 including the controls. These preparations were found ineffective against *F. hepatica* in cattle even in large doses, while in sheep they were unsatisfactory, killing some or all of the flukes in only part of the sheep treated.

A really satisfactory line of treatment was found in the use of kamala and its preparations. A preparation called Calbazan was found to have too little kamala for satisfactory results, but one called Parasiten was found satisfactory, as was kamala itself.

Marek states that a successful treatment for fluke disease has been demonstrated beyond question. By large doses of kamala all flukes can be removed from 85 per cent and a large proportion from 15 per cent of all patients. Either kamala depuratum or kamala venale is satisfactory. Yearlings and older sheep may be given 15 grams divided into two doses and given at 12 to 24 hour intervals. The entire 15 grams may be given in one dose to strong animals or it may be divided into 5 doses for weak ones. After treatment sheep are dull for 3 to 5 days, they lie down a great deal, eat little or may stop eating for a day or two, and have a diarrhea. The flukes die in from 3 to 8 days; the fluke eggs usually continuing to pass out until towards the close of the period. In cattle the results of treatment with kamala are beneficial but not so satisfactory as in the case of sheep; the dose used was 0.139 and 0.26 grams per kilo of weight of patient. Compared with kamala, male fern is less effective, more toxic, two to ten times as expensive, and of no value in the case of cattle. Male fern should be given to yearling sheep in doses of 5 grams a day for 6 days; for older sheep give 6 grams a day for 6 days; give in double this amount of some neutral oil.

Marek suggested the following plan for eradicating flukes from a farm: Treat all sheep and cattle at the beginning of stable life in the winter; for the next two weeks take all manure to a special place and cover with thick layers of manure from other animals to kill the eggs by rotting, or spread the manure on dry fields; repeat the treatment before putting the animals on pasture in the spring; if any animal shows signs of fluke disease put it up and treat as noted; if new stock is purchased, have the feces examined microscopically to determine the presence or absence of flukes.

M. C. HALL.

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DIAPHRAGMATIC RUPTURE IN DOGS. Harold Stanton, M.R.C. V.S. *Veterinary Journal*. 1st case. *From self-inflicted injuries*. Pomeranian, fourteen months old, was accustomed to jumping into the air at the instigation of his owner. In his last jump, he fell on the leg of a bedstead. He exhibited pains, appeared dull and got a dose of castor oil. Next day he refused food and began to vomit. Breathing was stertorous, and jerky. Auscultation revealed nothing on the right side but on the left there were exaggerated heart-beats and a peculiar gushing sound. Thirty-two hours after the accident the dog died. He showed at post-mortem a rupture of the dia-

phragm with the stomach, spleen and a portion of the small intestines in the left side of the thorax.

2nd case. *From External Violence.* Aberdeen terrier, 14 years old, was found in the gutter, collapsed and in great pain. He showed severe bruising of the ribs and left fore leg. After being eight days under treatment, he suddenly began to vomit, had labored breathing and died in an hour. At post-mortem there was found a ruptured diaphragm with the anterior portion of the stomach in the thorax.

3rd case. *Congenital.* A fifteen months' old Irish terrier had the following history: always with her owner and had never met with an accident. During his life his breathing had been peculiar, and he was breathless after a short run. He had a big appetite and his abdomen was much distended after a meal. He had pain sometimes. He would cough especially after feeding. Lately had fits of choking. It was while in one of these that he was observed to be gasping for breath and died.

Post-mortem: two floating kidneys. Stomach, intestines, spleen, pancreas and liver in the thorax. Stomach adherent to the diaphragm. Heart situated on the right side. Right lung half its normal size.

LIAUTARD.

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CONCERNING THE PFEILER PROPOSAL FOR THE COMBATING OF GLANDERS IN THE FIELD BY IMMUNIZATION. Dr. B. Schubert, Münster, i. W. *Copied from Berl. Tier. Woch. No. 30, July 29, 1915.* The earnest desire on my part to carry to victory a matter which according to my convictions was a worthy one, persuaded me—inasmuch as I am, on account of my official capacity in the glanders division of the Veterinary Biological Institute in Münster, i. W., a logical representative of the established methods for combating glanders,—to lay aside for once my induced and self-imposed reservedness and to take up a position with regard to the immunization against glanders in horses which while not new in itself is based on new standpoints with special insight into questions which have recently been brought forward.

If one obtains the very essence of the article which appeared in No. 28 of this periodical by W. Pfeiler on "A Proposal for Combating Glanders in the Field by Immunization" a prospect is arrived at which is certain to obtain the earnest consideration of specialists because he (the author) points to a practical way to attain the goal



which hovers before all those who have at heart the retention of the utility of our cavalry horses, namely the rapid freeing of the mobile and permanent horse depots from the danger of considerable losses through glanders. Whether one recognizes the grounds on which Pfeiler premises his proposal, as sound or unsound—granted that a complete eradication of glanders by malleinisation and serological examination is impossible, a concession to which I cannot agree from my own experience, and which would not be a convincing inducement, in the face of the present day standing of the disease of glanders, to take up other means of combating the disease which have not yet been proven trustworthy. The essential part of the proposal, vaccination of the animals threatened and also whenever possible the mobile cavalry depots similar to cholera and typhus vaccination, appears to me to be the true method and worthy of the warmest recommendation. The unavoidable contact of healthy horses with glanders infected animals which occurs under the extraordinary present state of affairs and which may be prolonged would be made truly non-dangerous or at least the danger would be considerably minimized.

The mallein ophthalmic test and the blood examination by means of agglutination and complement fixation can give in single cases as is most convincingly the case here in the West just as good a result, nevertheless the system involved leads very slowly to the point of freeing horse depots from the disease.

The distinct advantage of the process proposed by Pfeiler lies in the quick removal or diminution of the danger of infection—provided that the vaccination is carried out *en gros*. For, everything depends on the conservation of every single available horse. On this account the elimination of the horses sick with glanders to which principle of course Pfeiler naturally adheres, is indeed not more important than the protection from fresh infection even if it is not absolute and only relative.

It is not to be overlooked that the prevention of isolated formations through vaccination would be far less serious than in the case of the previous method where a quarantine of a week in the depots and forages is hardly avoidable.

A single diagnostic examination and simultaneously, with the removal of horses thereby recognized as sick, a vaccination of all the others left over would be sufficient to put a large depot in a position to move out. Should isolated cases transpire later, these can

be sorted out without danger to the healthy horses, the men also being vaccinated, which can also be done without danger.

That which Pfeiler outlines with regard to the application of diagnostic methods to the vaccinated horse I also subscribe to unreservedly. Even if the experiments instituted by him in this direction do not give any definite results, the expert would soon become practiced in sero-diagnosis or judgment of the mallein test even with altered and aggravated preliminary conditions, the inclusion of which according to my opinion is certainly demanded by the situation.

Apart from the material considerations which appear to make the immunization process in glanders possible throughout, none would deny high recognition of the act which Pfeiler has concluded in bringing his proposal into publicity. The effort that it must have cost such a vigorous and successful student and exponent of the hitherto existing "Nothing-less-than-elimination" methods to enter into the field of immunization publicly can only be most keenly understood by a worker in the same direction for nearly a decade.

May these lines help to direct the attention of those in authority towards the immunization process for glanders more than hitherto.

I am of the opinion that a rapid taking up of a vast wide reaching organization for the preparation of the vaccine and for the vaccination itself would very soon give results, which would be more advantageous to the fulfilment of our army's great tasks than the combating of the disease by the diagnostic methods, the apparatus of which has been rendered too cumbersome owing to the altered circumstances induced by the war. And also after demobilization, the complete or incomplete protection of the horses may exercise more of an opposing action against further ravages of a glanders epizootic in the civil population than the mere punctilious performance of diagnostic examination of the horses, which come to us from the land of an enemy.

I conclude: The execution of the Pfeiler proposals is to be warmly approved if it is carried out rapidly and *en gros* the great advantage thereof lies in that probably a materially bad effect is not to be feared.

REICHEL AND WERNER.

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ADENO-CARCINOMA OF UNDESCENDED TESTICLE. J. C. Lewis, D.V.Sc., B.Sc. *Australian Supplement Veterinary Journal*.—Five year old fox terrier had a hernia in the left inguinal region. Ex-

amination showed a firm, solid, almost immovable enlargement on the site of the internal abdominal ring. The swelling was not reducible. The denser part of the growth presented a movable attachment which was palpably not unlike a gut. In the scrotum, there was only one testicle present. The owner stated that a small swelling had been noticed a few months previously which had grown rapidly but gave rise to no general disturbance to the dog. Cutting down on the swelling, a greatly enlarged testicle was removed. The inguinal ring and skin were closed. The testicle was much enlarged and on section showed a firm new growth involving the whole tissue of the gland, which on microscopic examination proved to be an adeno-carcinoma.

LIAUTARD.

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A STUDY OF TUBERCULIN. A. Jousset. *Revue de la Tuberculose*, Vol. II, pp. 305-327, July 1915.—There is but one tuberculin, whatever be its botanical origin. In bouillon cultures there is spontaneously formed a part of the specific substance to which tuberculin owes its precipitant and local action in the tuberculous organism. Tuberculin is stable only in concentrated solutions. Its dilute solutions rapidly become unfit for the cutaneous test. Heating intensifies certain properties of tuberculin, rather than weakens them. Therefore, heat has a double action in the preparation of tuberculin: liberation of active substances by decoction of the bacillary bodies; intensifying the action of substances already in solution.

The active principle of tuberculin, or "tuberculous specifine" is derived from the cleavage of bacillary protein; it is associated with the cleavage product of nucleo-albumins. If this principle is a substance possessing chemical individuality, it is neither a proteose nor a peptone, but may be related to the amino acids. It is a substance of such elusive nature that it is almost chimerical to attempt its isolation.

BERG.

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SCAPULAR WOUND BY OBUS. Veterinary Major Darras and Briddee. *Bull. de la Soc. Cent.*—A chestnut mare, seven years old, had been wounded. Not entirely recovered she had on the right shoulder a fistula from which pus was escaping. She was in good condition and not lame. Probing of the fistula, which ran perpendicularly to the bone, revealed a rough bottom, but without the appearance of a foreign body. Free incision of the fistulous tract

and injection of antiseptic liquids formed the treatment. After a few days the condition of the mare had changed, she was very lame and the discharge of pus had become very abundant. For economical reasons the mare was destroyed. Post mortem: examination of the scapula showed on its superior third near the posterior border an ellipsoid perforation completely closed with fibrous tissue, except in its middle where there was a small opening from which pus was oozing. The posterior border of the bone was thickened and numerous bony deposits existed around the opening which, once removed from its fibrous tissue, had the aspect of a normal foramen. Under the scapula there was a piece of obus encysted in a mass of fibrous tissues.

LIAUTARD.

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LACERATION OF THE VAGINA IN A COW. A. Oeller. *Münchener Tierärztliche Wochenschrift*, Vol. 67, p. 47, 1916.—A few hours after parturition, a cow showed a large tumor-like swelling on the left vulva, as large as a man's head. Otherwise the cow appeared to be entirely normal and ate eagerly. The swelling seemed to be firm and almost painless. Temperature normal. On attempting to introduce the right hand into the vagina, a strong resistance was encountered. The owner informed me that the cow had calved without assistance and had cleaned herself. A second examination of the swelling and introduction of the index and middle fingers disclosed an abnormal opening in the left wall of the vagina. By pressing on the swelling with the left hand from the outside, something was pressed out through the opening which I could grasp with the fingers. By gentle traction the after-birth came out, followed by a quantity of fetal fluid. Since the after-birth had already been passed according to the owner, the only possibility was that it had become torn and only part of it was passed. The laceration of the vagina may have been caused by pressure of the forelegs or head against the left vaginal wall during severe straining.

BERG.

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VISUAL EXAMINATION OF THE BLADDER. J. C. Lewis, D.V.Sc., B.S. *Australian Supplement Veterinary Journal*.—The case is recorded to show the advantage that one may derive for the exact diagnosis of ailments of the bladder by the examination of the organ with the proctoscope. There is an illustration showing how the examination was made in the case of an aged draught horse suffering with incontinence of urine which was diagnosed as the result of



anatomy of the organ and was treated so as to give a comparatively satisfactory result. The case was specially presented to show that the examination of the bladder by this method will demonstrate clearly the nature of the internal aspect of the bladder and permit of a positive and sure diagnosis.

LIAUTARD.

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NYMPHOMANIA IN A 16 YEAR OLD MARE. George Wagner. *Münchener Tierärztliche Wochenschrift*, Vol. 66, p. 553, 1915.—In a 16 year old mare that had had 5 foals and which would not become pregnant, after several services, nervous symptoms appeared in the form of extreme irritability, striking and biting other horses or even people. This condition became aggravated during the course of 2 years to such an extent that the mare became a menace to life and had to be sold for slaughter. On the occasion of a severe attack, I was called by the owner, and found the animal in a state of highest excitement, dripping with sweat, continually pawing with the front feet and with these symptoms there were also grinding of the teeth and contraction of the abdominal muscles.

The administration of chloral hydrate and potassium bromide was without effect. Rectal examination disclosed hypertrophy of both ovaries. The owner objected to surgical treatment.

BERG.

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PETECHIAL FEVER AND ENDOCARDITIS COMPLICATED WITH ARTICULAR RHEUMATISM IN A HORSE. Mr. Nieder. *Rec. de Méd. Veter.*—A brown bay gelding, aged eight years, was brought to the writer suffering with high fever 39.6°C and 39.8°C and hyperleucocytosis (2000). The pulse varied between 48 and 59 a minute. Severe prostration, which suggested the idea of influenza, for which indeed the horse had been under treatment for a week. The diagnosis uncertain at first, was established four days after by the appearance of warm, diffused and painful swellings around the left fore fetlock. The myocardium was detected weak and the pulse went up to 90. The horse had myocarditis as a complication of the rheumatism. The endocarditis was not suspected as the beating of the heart remained clear and rhythmical although weak. Aspirin, in 100 gram doses, was given which lowered the temperature but did not affect the pulse. A few days later, the right knee was taken and also the posterior fetlock. The animal was in great pain and could scarcely stand up. Repeated doses of aspirin were followed

by an anorexia and complete adipsia. Salicylate of soda in 100 gram doses were given by the rectum with good results, the appetite improved but the pulse remained the same. The numerous petechial spots appeared on the nasal mucosa on both sides and on the lips. They disappeared with the injection of the serum of Jenser. The pulse was 100 and the heart beat was weak but clear. After a few days death took place with paralysis of the myocardium. The post-mortem revealed an ulcerative endocarditis, which had involved the entire tricuspid and a fibrous chronic endocarditis localized on the mitral. There was hypertrophy of the cardiac muscle and pulmonary edema.

LIAUTARD.

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A COMPARISON OF VARIOUS STRAINS OF THE *BACILLUS PSEUDOTUBERCULOSIS RODENTUM* (PFEIFFER), WITH SPECIAL REFERENCE TO CERTAIN VARIATION PHENOMENA. Shigeshi Kakehi, M.D., (Japan). From the Bacteriological Dept., Lister Institute, London, S. W. *Jour. of Path. & Bact.*, Vol. XX, No. 3, January, 1916.— Since the *Bacillus pseudo-tuberculosis rodentium* was suggested by Pfeiffer (1889) as the active cause of the pseudo-tuberculosis of rodents, a disease which had already been described first by Malessez and Vignal (1883-1884), and then by Ebert (1885-1886), and Pfeiffer's theory of its cause was confirmed by Preisz (1894), the character of this organism has been exactly investigated in detail from all points of view by a great number of authors (cf. Poppe's monograph on "Pseudotuberkulose," 1913).

SUMMARY. 1. Several strains of the *B. pseudo-tuberculosis rodentium* (Pfeiffer) dealt with are quite aerobic, make broth more or less turbid for a certain time, and turn litmus milk alkaline within five days. Further, when they are inoculated in various carbohydrate liquid media, they show all the properties already well-known, and, in addition, a slow acid-forming action is invariably seen with adonite, this being readily accelerated by sub-cultivation.

2. When various liquid media inoculated with this organism are incubated for some time and then sown on agar plates, we obtain in every case three kinds of colonies. One of these kinds (A-colony type) possesses the characteristics of the usual type of colony of this organism, namely, an almost transparent, slightly bluish shimmer, while the other (B-colony type), which must be considered as a variant form, has a greyish-white, opaque appearance. The third form (C-colony type), which must be considered as a transition stage between A- and B-colony, consists of a greyish-white, opaque centre and an almost transparent marginal zone.

3. A-colonies, the genotypical form, when repeatedly sown on agar plates, always produce only the same kind of colonies. B-colonies, on the contrary, upon further sowing return to the genotypical form in certain generations by means of forming the third form, C-colonies. The peripheral part of these always produces A-colonies, while the central part gives birth to the variant form, B-colonies.

4. The bacilli in B-colonies are definitely distinguishable from those in the genotypical form by some slight morphological differences and by the fact that they keep broth quite clear from the beginning and acquire a high viscosity in association with high agglutinability, as well as by showing more slowly their characteristic action on adonite.

5. Some strains of this organism, when repeatedly cultivated on agar in the ordinary way, acquire to some extent at a certain stage, properties resembling those of B-colony bacilli. They show a high viscosity combined with high agglutinability, almost lose the property of making broth turbid, and their action is sluggish in fermenting adonite, while in other points there is no distinctive difference. Those strains which have been cultivated in various liquid media are much more liable to produce variant forms.

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REICHEL.

FORMOL IN SEVERE CASES OF DISTEMPER. Veterinary Major Chouleur. *Bull. de la Soc. Cent.*—Wishing to experiment with the method of Bottini in the treatment of gangrenous pneumonia, the author has resorted to it with several young horses affected with severe distemper and having intra-thoracic localizations. Bottini's treatment consists in the intratracheal injection of 10 c.c. of a solution of formol at 4%. The technic is simple. The anterior face of the trachea, at its superior third, is shaved and disinfected. With a fine trocar sterilized and a Pravaz syringe 10 c.c. of a solution of 4 grams of formol in 100 grams of distilled water is slowly injected. No secondary accidents follow, except that in some animals short spells of coughing occur. It is essential to resort to this injection as early as possible on animals which show alarming symptoms and before material lesions have taken place. When applied at the proper time, the formol acts by its antithermic properties and the temperature which had remained above 40°C for some time will, after the second or third injections, drop to normal. The antiseptic properties of the drug will also have a powerful influence.

LIAUTARD.

## PROCEEDINGS OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

*(Continued from page 431)*

DR. R. C. MOORE: It seems to me we will have to modify our minutes a good deal on this morning's work. If I remember correctly, Dr. Dunphy's motion was that the members of this committee be elected by the association. Finally that motion was amended and accepted by Dr. Dunphy that a committee of three be appointed to select a group of men from which to select or elect these members.

DR. KINSLEY: There is another motion on record providing that there were to be five on this committee, one to retire each year, and a new man appointed for five years each year.

DR. DUNPHY: Was that resolution put and recorded in the record?

DR. KINSLEY: Yes, put and carried.

DR. KNOWLES: If an amendment could be entertained to this motion now, I would like to move to amend the motion to the effect that the minutes of this morning with reference to the appointment and election of the committee shall read, instead of five years, seven years, with one member retiring each year. I will offer that as an amendment to the motion.

DR. MAYO: This can be better accomplished by simply accepting the report of the committee and having the resolution read that they accept the committee's suggestion of seven members instead of five. That is all that is necessary. Then go on and provide that a new member shall be elected each year. They will automatically replace themselves. I also think this resolution should carry with it an amendment that the number suggested by this committee be appointed by this association that appoints the committee at the same time.

PRESIDENT MARSHALL: Are you ready for the question? All those in favor of accepting the recommendation of the special committee make it manifest by saying "aye"; those opposed "no". The ayes have it. It is so ordered.

DR. S. STEWART: I move that these gentlemen be elected to this memorial committee. Seconded by Dr. Kinsley.

PRESIDENT MARSHALL: You have heard the motion, that the seven gentlemen named be elected as members of this committee. All those in favor of their election make it manifest by saying "aye." Opposed "no". The ayes have it. It is so ordered. I would like to ask a question in reference to Dr. Winchester,—I wrote him asking him to act as chairman of the committee and I think Dr. Hoskins wrote him. He said he could not act on any committee. It may be he refused because he did not intend to attend, or it may be that he is not physically able to act on the committee.

DR. KNOWLES: Whenever there is a vacancy the chairman can fill it.

DR. HOSKINS: I think Dr. Winchester's feeling was that he could not accept the chairmanship or secretaryship. He has done an enormous amount of work on this committee.



DR. V. A. MOORE: I am sure you are all very anxious that the committee shall be an efficient one, and we all know the good Dr. Salmon did for the veterinary profession. He made constant and persistent effort to uplift it in every way possible. The suggestion I have to make is this: that the committee reported by Dr. Hughes be composed of seven instead of five members, because we feel that seven would be more efficient than five. For one reason, we wished to get these people distributed over the country a little better. The point is, we feel very keenly the difficulty this committee is going to have in circularizing and getting at the individuals who may be willing and glad to contribute money. The New York State Veterinary Society at its meeting the first of August appointed a committee for the purpose of collecting funds for this memorial from the New York people, and the committee was instructed to co-operate with the committee appointed by this association. I think if, when we return to our homes, each member would seek to interest his own state society and get it to appoint a committee to collect funds from members in that state, men belonging to the society first perhaps, and then those not belonging, and then if the committee, just suggested, should communicate with the chairmen of these state society committees, we would have the entire country covered. Every practicing veterinarian in the country would have a chance, or an invitation (for I would consider it a privilege) to contribute to this fund. It would hasten matters very materially, and I believe would help us to get a much larger sum than by the solitary efforts of this central committee.

DR. KNOWLES: Dr. Moore's proposition reminds me of one thing. I am secretary of our association in Montana. I think one way in which this memorial may be brought to the attention of the veterinarians is by placing a circular letter, with all the members of the profession in the state, explaining clearly the object and the system and the method by which this is to be carried into effect. Whether each state shall formulate such letters, which must of necessity be of considerable length, or whether this memorial committee shall furnish such circular letter is a matter of detail to be decided later. If this matter could be taken up by each of our associations and talked over with the members, it will stimulate interest. When it comes to reaching those who are not in the associations, it would take a written request, for a contribution to the Salmon Memorial Fund, containing sufficient information to show what we are trying to do. This would appeal to a good many persons, and I believe it would bring more money from people who would not otherwise contribute.

DR. R. C. MOORE: How do we know, how does the committee know, which members are elected for one year, and which for seven years?

DR. KINSLEY: Let them settle that.

DR. HUGHES: I do not know whether the committee's work ceased with the presentation of that report or not. I might mention that the committee arranged the names in the order in which the committee thought it should be done.

PRESIDENT MARSHALL: Are you willing to leave it to the committee to settle it for themselves?

DR. JENSEN: Leave it to the committee.

PRESIDENT MARSHALL: Is there anything else to be said on this subject? Dr. Campbell just told me that he learned of one other deceased member since he read his report. That is Dr. R. R. Hammond, of Chetopa, Iowa. The next order of business is the report of the committee on resolutions by Dr. Shepard.

Whereupon Dr. Shepard read the report of the committee as follows:

Whereas, through the foresight and generosity of John D. Rockefeller, by means of the Rockefeller Foundation, an Institute for research in diseases of animals has been established, and its maintenance provided for in our country, which is certain to be of immense value in the conservation of the livestock interests not only of the United States but of the entire world:

Therefore, BE IT RESOLVED, that the A.V. M. A. recognizes its value and appreciates the generosity of Mr. Rockefeller.

DR. KINSLEY: I move the adoption of the resolution. Seconded by Dr. R. C. Moore.

PRESIDENT MARSHALL: It has been moved and seconded that the resolution be adopted. All those in favor signify by saying "aye"; those opposed "no." It is carried.

DR. SHEPARD: Whereas, the livestock interests of this country have recently been menaced by that fatal animal plague, foot-and-mouth disease; and

Whereas, the methods adopted by the Bureau of Animal Industry, United States Department of Agriculture in the control and eradication of this disease, have succeeded in practically wiping out the infection:

Therefore, BE IT RESOLVED: That this association express its hearty commendation of the methods employed by the Bureau of Animal Industry in its monumental task of circumscribing the area of infection as much as possible and thereby aiding in the protection of other sections of the country which otherwise must have shared in the general infection; and

BE IT FURTHER RESOLVED: That in the opinion of this association the gratifying results obtained could not have been secured in the time by other means than those employed, nor under other authority than the Federal Bureau of Animal Industry as at present constituted.

DR. KINSLEY: I move the adoption of the resolution. Seconded by Dr. R. C. Moore.

DR. TORRANCE: Mr. President, I would like to call attention to the use of the word "fatal" in describing the disease. I think you are all aware that

the disease is not fatal. I would like to have the word changed, substituting therefor, the word "serious".

PRESIDENT MARSHALL: Dropping the word "fatal" and using instead the word "serious", are you ready to recommend the adoption of the resolution? Those in favor of the motion make it manifest by saying "aye". Opposed "no". The ayes have it. It is so ordered.

DR. SHEPARD: Whereas, the 51st annual meeting of the A. V. M. A., held in Oakland, Calif, has proved an unqualified success, due to the admirable location and other local features, but especially to the splendid efforts of the various local committees, and the hospitality of the good people generally:

THEREFORE, BE IT RESOLVED: That the sincere thanks of this association be extended to all who in any way contributed to the success of the meeting: and

BE IT FURTHER RESOLVED: That special thanks are due the press of Oakland for the large amount of space devoted each day to the deliberations of the association; and

BE IT FURTHER RESOLVED: That an expression of appreciation and thanks is hereby tendered the management of the Hotel Oakland for its efforts toward the success of the meeting.

Signed by Drs. Dalrymple, Nelson, Norton, Rutherford and Shepard, chairman pro tem.

DR. KINSLEY: I move the adoption of the resolution. Seconded by Dr. Hoskins.

PRESIDENT MARSHALL: All those in favor of adopting the resolution signify by saying "aye"; opposed "no". The ayes have it. It is so ordered.

DR. HOSKINS: I move that the report as a whole be accepted. Seconded by Dr. Kinsley.

PRESIDENT MARSHALL: It has been moved and seconded that the report as a whole be adopted. All those in favor of the adoption of the report signify by saying "aye"; opposed "no". The ayes have it. It is so ordered.

Has anyone else a resolution to be adopted?

DR. STEELE: Mr. President, I did not hear the names of the speakers at the banquet last night. I think they were Mr. Hunt and Mr. Lively. I think these men should be recognized by a communication from the A. V. M. A. with thanks. I make a motion to that effect. Seconded by Dr. Hoskins.

PRESIDENT MARSHALL: It has been moved and seconded that a letter of thanks be sent to Messrs. Hunt and Lively for the interesting addresses they gave us last evening. Any remarks? If not, those in favor manifest it by saying "aye"; those opposed "no". The ayes have it. It is so ordered.

There are two or three other matters on the program to be called for. The glanders committee, for instance, that committee has made no report. There is

no report from Dr. Milks, the delegate appointed to the American Pharmaceutical Association. That completes the program with the exception of the special business for which the meeting was called.

DR. NEWSOM: Owing to the fact that the report of the committee on veterinary anatomical nomenclature was not published and it was not deemed advisable to recommend its adoption at this meeting, I want to give notice that after it has been published, I presume a year hence, that motion will be forthcoming, so that you may be thinking about it.

DR. MAYO: I do not understand the Doctor's statement. The report of the committee has been presented and accepted by the association. I have the report in my possession now.

DR. KINSLEY: When was it accepted?

DR. MAYO: I say, it was presented to the association.

PRESIDENT MARSHALL: I think it was accepted. That is the term which was used.

DR. NEWSOM: I did not understand that that carried with it the adoption of all those names by this association. I am not complaining if that is the construction put upon it.

DR. MAYO: I understand that the association has accepted this report and has accepted those names because it is practically all names and nothing else. If they accept it they accept the names.

DR. MURPHEY: I was unable to be present when this report was submitted, but I have attended previous meetings where a report of this kind has been submitted, and I believe there is a feeling among some of the teachers of anatomy that it is not fair to adopt this until we have had some chance to examine it in detail. There may be some difference of opinion, not only in regard to terms but to construction which should be settled one way or another before we finally adopt them. I believe there was a committee appointed at the New York meeting to prepare this report and the committee prepared a report and asked for the publication of it so that it might be submitted to the schools and thus get some concerted action before this meeting. I am informed that the secretary of the association refused to honor the bill for the publication and refused to allow the report to be submitted to the various colleges, as was the intention. I do not believe it is fair to consider it until we have had a chance to examine it.

DR. S. STEWART: An acceptance of a report is not the adoption of the report. This is a very important matter, and there has been a considerable number of people interested. If this association adopts the nomenclature submitted by this committee, then it becomes an official statement issued by this body. I am in hopes this body will do so. It ought to do so, fully understanding what the adoption of the report signifies. I am glad Dr. Newsom has given notice that one year hence he will move the adoption of that report.

DR. MAYO: The report of the committee as has been fully explained to the association, was presented at the New York meeting, or at least a portion of the report was presented. All that had been completed up to that time was accepted and published in the official proceedings of this association and is a



part of the official minutes. We have already adopted that system of nomenclature,—all we could. The additional report of that committee was furnished to me, and as there were no funds in the treasury to pay for republishing what had already been published—over sixty pages of typewritten anatomical names, I did not think it wise to draw a voucher on an empty treasury, I therefore referred the matter to the executive committee for their action, and in so far as I was responsible for presenting this matter to the executive committee, I assumed that responsibility. The executive committee, however, voted under the conditions, not to publish that and send it to all members of the association, which would have been an expense of about two hundred dollars or more.

DR. MURPHEY: Do we consider that this has been adopted officially, or do we not?

PRESIDENT MARSHALL: My opinion is that accepting the report does not mean endorsing it. If we wish to make that the official standard of this association we must do something more.

DR. CAMPBELL: That brings us back to the question that came up before. About the glanders report—I notice in the treasurer's report, circulated here, there are a number of expense accounts for that committee. Is that for the present report that has not been submitted, or was that in payment of the expense in connection with the report of the last year?

DR. MAYO: I think that was for the expenses incurred previous to the New York meeting. I think these bills were held over and paid, as I recollect it now, since the last report. I have drawn no vouchers for this present committee. I could tell by looking at my books.

DR. CAMPBELL: I merely wanted to move that the committee be given a further chance to get up their report and hand it in, as we had already spent some money on it, but since we have had our money's worth, I will not make any motion.

PRESIDENT MARSHALL: If there is no further business we will hear the report of the executive committee up to date.

DR. MAYO: Unfortunately the stenographer who took the report this morning has not his notes written up and I have not been able to find him. I can tell you, however, that the executive committee recommends that the list of accepted colleges published in the 1913 report be accepted as the list of accredited colleges for the coming year. Ontario has already been accepted by special vote this morning.

DR. HOSKINS: I move its adoption. Seconded by Dr. Kinsley.

PRESIDENT MARSHALL: It has been moved and seconded that we accept the report of the executive committee with reference to the accredited list of colleges. Any remarks? If not; all those in favor of the motion signify by saying "aye"; those opposed "no". The ayes have it. It is so ordered.

DR. MAYO: The recommendation of the committee on colleges that the general plan of submitting the committee report in two sections, one for reading and publication; the other a supplemental report consisting of criticisms and suggestions prepared by the committee for transmissal by the association secretary to the college concerned, be approved and continued.

DR. KINSLEY: I move that the recommendation of the committee be adopted. Seconded by Dr. Hoskins.

PRESIDENT MARSHALL: It has been moved and seconded that the recommendation of the committee be accepted. All those in favor of the motion signify it by saying "aye"; those opposed "no". The ayes have it. It is so ordered.

DR. MAYO: The recommendation of the committee on colleges which would provide for a re-organization of the committees on intelligence and education and on college investigation. I will read that part of the recommendation: "We would again call attention to the overlapping of the committee on college investigation and the committee on intelligence and education, and we again recommend the adoption of some plan similar to that of the American Medical Association, a fairly stable council involving the inclusion of these two committees. We propose the following by-law amendment: Substitute for the 5th line of article 7 the following: Committee on Veterinary Education of 3 members, term of office 3 years, and one membership expiring each year. Substitute for section 2 the following: It shall be the duty of the committee on veterinary education to investigate the work of the American Veterinary Colleges, and to report to the association such general findings, suggestions and criticisms concerning the same as it may deem advisable. It shall also be the duty of this committee to annually recommend to the association a list of veterinary colleges for recognition by the association with reference to eligibility to membership of their graduates. It shall also be the duty of this committee to keep in touch with the general progress of education in the veterinary and allied professions, and make such report from time to time as it may deem advisable."

It is understood that it would be recommended to the incoming president to consider this question.

DR. HUGHES: There are several members of the committee on intelligence and education present. Is it the idea to pay their expenses incurred in making these examinations?

DR. MAYO: I would say in answer to that, that generally not over two members of the committee visit a college, and sometimes only one of the regularly appointed committee, and a member of the association is appointed from that immediate neighborhood to make the examination. If it is a matter of some distance where the travelling expenses are great, only one member of the committee has gone, and a temporary member of the committee has been appointed to assist in making the inspection. This temporary member being a resident of the immediate locality, it means the saving of considerable expense. It is the plan of the executive committee that this custom shall be followed with reference to the committee on intelligence and education.

This arrangement would give five members scattered over a wider territory, so that one member of the committee at least will have an opportunity to visit a college.

DR. S. STEWART: Do I understand that the purpose of this recommendation is to do away with one of the committees, and that by voting in the affirmative we can dispense with one committee and add the duties of the other committee

to it at the same time? In other words, whether a motion to that effect can accomplish that end? Personally, I should be glad to see the two committees merged but I am not sure but three members on that committee would do more work than five. Oftentimes one man with power to call in help, can accomplish the same thing. I think three are sufficient.

DR. MAYO: The veterinary college investigating committee is a committee of three. It was the plan of the executive committee to assign their duties to the committee on intelligence and education. It was understood that they would have a sub-committee of three, or use their best judgment in regard to that.

DR. DUNPHY: When this committee was organized or authorized by the association, there was a certain sum, \$500 I believe, set aside to defray the expenses. There was no regular rule laid down by any by-law, under which this committee should operate. We met in Chicago and elected Dr. Tait Butler president at that time, and we formulated a certain regulation that we should follow in this work. We found that the funds were not sufficient, unless we divided up the work, and even then we could scarcely reach all the colleges. We divided the work: one member was to investigate a certain college, and the other members of the committee agreed to have some local man in the locality who was not connected in any way with the college to make the second member of that committee to investigate the college. We carried out these regulations. That is, in talking it over with Dr. Reynolds, the chairman of the committee, we decided that the association should have some regulations in the by-laws whereby the duties of this committee should be regulated and we believe, owing to the overlapping duties of the two committees, it would be better to combine them in some way, if the committee saw fit.

DR. HOSKINS: I move the adoption of the recommendation. Seconded by Dr. Jensen.

DR. MARSHALL: Does this mean we are recommending an amendment to the by-laws?

DR. HOSKINS: No, the special committee is not provided for by the by-laws. The special committee retires, as I understand it, and the regular committee fixed by your by-laws takes on the duties of the special committee.

DR. S. STEWART: I would like to know whether or not this committee would have any special funds to do anything with, or whether they will be on the same basis as they are at present?

PRESIDENT MARSHALL: I presume that would have to be taken care of if we dispose of this question. Are there any remarks with reference to the adoption of the recommendation?

DR. MAYO: I will say that in regard to Dr. Murphey's suggestion on the report of the committee regarding provisions in the by-laws, that as the constitution and by-laws were being revised it was thought by the committee that that recommendation would be taken care of in the revision.

PRESIDENT MARSHALL: All those in favor of accepting and adopting the recommendation, make it manifest by saying "aye"; those opposed "no". It is carried. It is so ordered.

That finishes the report of the executive committee.

There have been no appropriations made for the various committees. Shall we take that up now, or wait until this evening? It is called for now, so if there is no objection, we will proceed with the matter of appropriations. Do any of you know what will be wanted for these committees?

DR. S. STEWART: I move that the committee on intelligence and education be appropriated \$500 or such portion thereof as may be needed to carry out the work of the committee.

PRESIDENT MARSHALL: Was there a recommendation made by the executive committee that all recommendations for appropriations be referred to the finance committee?

DR. MAYO: No. That recommendation was made in the secretary's report, because sometimes toward the close of the session, in the hurry of closing, resolutions are put through appropriating money without due consideration, and in the low state of the treasury it might be rather embarrassing for your secretary to have to hold up appropriations which were made by this association. It was not because I was not in favor of publishing the reports of the Committees. It was simply because we did not have any funds to publish them with. For that reason, I recommended that all resolutions appropriating funds should be referred to the finance committee. I did not think it would make any difference if they were referred to the executive committee, but the finance committee hasn't much to do, and I did not want them idle.

PRESIDENT MARSHALL: It seems rather important that we have some check on the appropriations. Each of you connected with a committee is (and very naturally too) exceedingly anxious to have money enough to do the work and do it well, and with your anxiety you frequently go beyond the amount we can give you.

DR. HOSKINS: I trust when these resolutions are passed or appropriations made, it shall be considered whether that money is actually set aside. I ran up against a situation where there was \$1000 appropriated in 1912 for the entertainment of any delegates from the countries of continental Europe. That \$1000 was not utilized at all because there was no one who came over from Europe. That \$1000 had been set aside in 1912 to be used in 1913. I went before the association and asked that that \$1000 which had been wisely appropriated and set aside, should be turned over to the legislative committee to continue its effort in behalf of the Army Veterinary Service Bill and to look after such other legislation passing through Congress as might affect the interests of the profession, and it was done. But I was not able to get that money until recently—some time in May I got the balance of the money. It wasn't much of a hardship but it happened frequently that the committee was in my personal debt two or three hundred dollars.

DR. MAYO: No one is more anxious than I that every bill shall be promptly paid and in the case of every bill that has been presented to me with one single exception, a voucher has been drawn and forwarded through the proper channels immediately to the treasurer. Those of you who know Dr. White know that at the previous meetings he has sat on the lid of the treasury and has repeatedly called the attention of this association to the fact that they were



appropriating monies when they did not have any to appropriate. As a matter of fact, when the proceedings of the last meeting were published, the association was \$3000 in debt. They could not set aside \$1000 unless they borrowed it. We borrowed the money to pay the indebtedness. Whether the treasurer has set aside sums for these appropriations, I do not know, but I don't think he has, because he did not have it to set aside. It is my impression that he has paid the most pressing bills of the association, but those who are long-suffering and good natured, like Dr. Hoskins and some others, have been put off.

DR. HOSKINS: I am not complaining, but it seems to me we ought to be able to calculate what our expenses are, and there should be some restraint put on committees. When money is once appropriated, it should be set aside to cover the appropriations for those committees so that they will not be hampered. In the case of the legislative committee we were not hindered or disturbed in any way. We went right on with the work. It had to be done at that time, and we did it.

PRESIDENT MARSHALL: The motion has been made by Dr. Stewart. Was it seconded?

DR. KINSLEY: If it wasn't seconded, I will. I second the motion.

PRESIDENT MARSHALL: The motion has been made by Dr. Stewart and seconded that the association appropriate for the use of the committee on intelligence and education a sum not to exceed \$500. Any remarks?

DR. CAMPBELL: I want to move an amendment of that motion. I have heard other members of the committee state that they would like to bring the appropriation for this committee up to such a sum as would enable them to visit all the schools in one year. I know it has been the custom to visit one school one year and another school another year, and others at another time. I have heard criticisms of the committee because they could not visit all the schools in one year. It is a hard matter to make comparisons of the different schools when the visits are so far apart, and conditions may be different one year than another. I am sure the work of the committee could be done more efficiently if the appropriation were sufficient to make it possible to visit each school in a year. Now, if you limit the appropriation to \$500 they will not be able to visit the schools all in one year. It will take them nearer three years to make the rounds. I would like to amend that motion to read "an amount sufficient to permit at least two members of the committee to visit all of the schools published in our accredited list and any others which might make application for listing on the accredited list in one year."

PRESIDENT MARSHALL: Have you any idea how much that would amount to?

DR. MCCAIN: That brings up the very point we have been discussing, in the matter of appropriations. I am afraid if the appropriations are going that way from the house to the finance committee, it will lead to a great deal of trouble. It has occurred to me to offer the suggestion that the chairmen of the various committees which require appropriations for their work should communicate their desires at once to the finance committee, and let that committee report back to this association with recommendations which can be adopted or rejected as you see fit.

DR. KINSLEY: As the finance committee is listed here simply for the purpose of examining the books, and their duties are explained and limited by the constitution and by-laws, I think that will complicate matters more. It seems to me, under the circumstances, if any reference is to be made, it should be made to the secretary and then submitted to the executive committee. At the present time we have to look after, first of all, the journal. I don't know whether these gentlemen have determined the amount of money demanded for that. At any rate, they will have a better idea of the financial standing of this organization than anyone else. I do not see how it will facilitate matters by referring it to the finance committee.

DR. R. C. MOORE: As this organization is largely for the benefit and the up building of veterinary knowledge and veterinary science, I believe that our educational interests are one of the first things to be considered, and to do that, we must naturally and inevitably spend some money. I believe great good has come from the work of this committee, and I believe it should be fostered. Therefore, I will offer as an amendment to Dr. Stewart's motion that this be made \$1000 instead of \$500.

DR. DUNPHY: I see a great deal of force in what Dr. Campbell has said in regard to the work of the committee investigating colleges. The first year the committee had to divide its work up and at the same time they had to postpone their visits to some colleges for another year. It was fortunate that we had two members with the Bureau of Animal Industry who were going to California on official business, and they made some of the investigations, thus saving us some expense. Then those two members that were appointed by the Bureau of Animal Industry went to Terre Haute, Indianapolis, Cincinnati, and Columbus, and I went with them. That saved the expense of another member of our committee going. When we reached Washington Dr. Eichhorn kindly assisted me with the colleges there. If it had not been for that, we would not have been able to do as much as we did. But for those fortunate circumstances we could have done little in the inspection of colleges on the amount assigned us. Further I would say that being a member of the committee for three consecutive years, I could see that there was a wonderful change on the part of the colleges owing to the fact that they had been investigated by the committee and expected to be investigated from year to year. They had taken advantage of friendly criticism offered by the committee and had improved their institutions in a large measure. I think Dr. Campbell will bear me out in that statement. He was associated with Dr. Reynolds this year in inspecting some of the colleges and Dr. Reynolds no doubt has told him how we found these colleges when we first started in on the work.

DR. MAYO: In appropriating \$1000 a year for this work, I want to call attention to a fact which should be borne in mind, and that is the resolution providing for an amendment to the by-laws raising the dues to \$5 a year does not go into effect until a year from now, and you are now running on the \$3 a year dues basis. You have something over \$3000 in the treasury at the present time—(probably not that much when the outstanding bills are paid; I am sure it is not over \$2500,) to run this association for the coming year. Don't forget that. Do not think I am trying to cut these committees down—

I would not for a minute—I would give them \$2000 if we had it to give. It is easy to appropriate money, but those of us who have to pay it out when we haven't it, know how embarrassing it is. It brings this association into disrepute to have to hold its bills for printing and things of that sort until we can get enough money in the treasury to pay them. It is not right.

DR. S. STEWART: I would like to modify the amendment of Dr. Moore and my motion. Instead of "the appropriation shall be \$1000 or as much thereof as shall be needed", change the wording thereof to "as much thereof as may be available" and leave it that way so that the secretary and the president and this committee may decide."

DR. KINSLEY: I accept that modification.

DR. MAYO: I will not assume that responsibility.

DR. HOSKINS: I think this is very unfair to the incoming president or the incoming secretary to put that burden on them. I know how it will be. The committee will do the work; the bills will go to the secretary who will draw vouchers as promptly as they are called for; the vouchers will go to the treasurer; and if the money isn't at the other end of the line, the voucher will just stay there. I think it is very unfair. Then again think of the discord it will create among the committees. If I get the money and the other committee does not, there would be criticism of the officers and I would be accused of having a pull.

DR. MAYO: I just want to call your attention to the position such a measure would put us in. For instance; this association votes \$1000 to any one committee and the chairman of that committee immediately puts in a bill for that \$1000. We will say three committees have \$3000 appropriated, and put in a bill for the full amount of the appropriation and I draw a voucher, and it is taken out of the treasury. Then what are you going to do?

DR. S. STEWART: But if the appropriations are made by the officers who are in touch with the situation, pro rata, are they not the best persons to make appropriations, because they have the knowledge to do it with?

DR. NEWSOM: Mr. President: I believe that in the appointment of this committee the president could take into consideration the location of the various colleges and so arrange the location of the five members on this committee that the investigation could be easily done on \$500. Speaking from the standpoint of our school I will say we would like to have all five members of the committee visit us. But as that seems to be impossible under the circumstances, we will be satisfied with one, and I believe \$500 would be sufficient for this year.

DR. CAMPBELL: I want to make myself clear to some who may think I am trying to see money going out of the treasury. I think this association has never done any work that is more important than the work done by that committee on college investigation, and I think there is no other work that needs to be done more thoroughly from what I know of the work. I would like to see the association appropriate enough to do it thoroughly, but if funds are not available, then let the committee take longer to do it. I would rather see it done thoroughly once in two years, than to have it done half way in one year. Do it thoroughly or not at all.

DR. ELLIS: From the reports we have received from year to year and especially this year from the committee on college investigation, I think they

have accomplished a great deal on that \$500 and I think it would be very unwise to cease operations. I would say, do what you can with the \$500. They have done wonders. Continue on that line until you can appropriate the \$1000 or \$2500 if you have it.

PRESIDENT MARSHALL: The question is called for. May I speak a minute and not put the question? Our funds are limited, and we have not heard from the other committees. I think we ought to find out what is wanted by all. What is the use of making appropriations if we haven't the money to meet them with? I am seriously in doubt whether we can afford to allow this committee \$500. It seems to me it would be better if the association had an appropriation committee which would examine into all these matters, hear the different committees as to their wants, and then recommend to the association what, in their best judgment, can be given, and let the association consider the recommendations. I am ready to submit the question if there are no further remarks.

DR. KINSLEY: How would it be to have the president, secretary and treasurer compose an appropriation committee? The two men at least, and the third being the president, all would be in close touch with the organization and would know what could be appropriated. It seems to me this would be the best way.

DR. MURPHEY: I move a substitute motion; that the finance committee be appointed and the Babson system adopted.

PRESIDENT MARSHALL: There is a motion before the house and unless the movers and seconders are willing to withdraw their motions, I cannot entertain your motion.

DR. KINSLEY: Isn't that a change in the by-laws anyhow? It would have to lay over a year.

PRESIDENT MARSHALL: What will we do with the original motion?

DR. S. STEWART: The original motion seems to stand before the house with Dr. Moore's modification.

PRESIDENT MARSHALL: That was not seconded.

DR. S. STEWART: Undoubtedly it would be absurd for this association to make appropriations for this committee next year if our secretary feels sure the bills already incurred and unpaid would not allow any appropriation. Better not spend money before we get it, and I have felt that we might correct such a possible condition of spending money before we got it by leaving it with the secretary, treasurer and president, and let them be a committee to determine whether those funds are really available or not. We want to know, when the meeting is over and the bills are all paid, how we stand. There may not be a dollar left for our committees, after the indebtedness of establishing a journal along the lines contemplated are met. There may not be a dollar available. The committee ought to know whether there is any money before they ask for appropriations. Is it available? The secretary and treasurer can advise us if there are any funds. That was my idea in making the motion, and I made that as a modification, subject to the direction of the president and secretary as to the availability of the money.

DR. KINSLEY: If I seconded the original motion, I will accept that modification.

DR. MAYO: I think the treasurer ought to be on that committee.



PRESIDENT MARSHALL: The president, secretary and treasurer, is that the way you understand it?

DR. MAYO: Just a word before that motion is put. You know that every committee thinks its own work is the most important work of the association, and properly so. I think that a committee, to be efficient, must have that spirit. Now when the present secretary or rather the executive committee took this matter in hand this year, I know that the committee felt hurt and they had cause. The committee's report was one of the best committee reports that has been presented to this association, but the executive committee with the secretary did not feel that the publication of that report and the expenditure of the money was as important as some other things. It is for you to decide.

DR. R. C. MOORE: I would like to call attention to the fact that the committee is appointed to investigate these colleges and is asking for the necessary funds. Does not the association ask the college committee to do this work! Should not the committee be supplied with funds so that they can work?

PRESIDENT MARSHALL: Are you ready for the question?

DR. FROTHINGHAM: What is the question?

PRESIDENT MARSHALL: I will ask Dr. Stewart to state the question again.

DR. S. STEWART: It is moved that we appropriate the sum of \$500 or less as may be needed, by the committee on investigation of colleges, subject to the findings of the appropriation committee, composed of the president, secretary and treasurer, as to whether that sum is available.

PRESIDENT MARSHALL: You have heard the motion as stated by Dr. Stewart. All those in favor signify by saying "aye"; those opposed "no". It is carried. The motion is unanimously adopted.

DR. HOSKINS: I would like to ask for an appropriation of \$500 for the legislative committee. I hope ultimately an important work will be done. I feel confident Congress will pass this bill and I offer that as a motion because a great deal of this money has already been expended. There will be a new Congress and the work will go on just the same. In December, of course, Congress will meet and the expense then will accrue very much faster. I want to say that the army veterinarians are co-operating with our committee in the matter of work and money and the utmost of their time.

PRESIDENT MARSHALL: Will you accept this appropriation under the conditions which Dr. Stewart has stated?

DR. HOSKINS: Yes.

DR. KINSLEY: I second the motion.

PRESIDENT MARSHALL: The motion has been made and seconded that \$500 be appropriated to the legislative committee under the same conditions stated by Dr. Stewart in moving the appropriation for the college investigation committee. Any remarks? Those in favor of the question make it manifest by saying "aye"; those opposed "no". The "ayes" have it. It is so ordered.

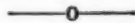
The time for adjournment has arrived. The session to-night for the election of officers is scheduled for 7:30. The election of officers is the only thing left. We will begin promptly at 7:30. If there are no objections we will stand adjourned until that time. That means 7:30. Be prompt, gentlemen.

*(To be continued)*

## MEETINGS OF THE ASSOCIATION

1863. First Meeting, New York, N. Y., June 9 and 10.  
1864. Semiannual (comitia minora)—New York, N. Y., Jan. 19.  
Annual—New York, N. Y., September 6.  
1865. Semiannual—New York, N. Y., March 7.  
Annual—Boston, Mass., September 5.  
1866. Semiannual—New York, N. Y., March 5 and 6.  
Annual—New York, N. Y., September 4.  
1867. Semiannual—Boston, Mass., March 5.  
Annual—New York, N. Y., September 3.  
1868. Semiannual—New York, N. Y., March 5.  
Annual—Boston, Mass., September 1.  
1869. Semiannual—Boston, Mass., March 16.  
Annual—New York, N. Y., September 21.  
1870. Semiannual—Philadelphia, Pa., March 15 (no quorum).  
Annual—New York, N. Y., September 20.  
1871. Semiannual—Boston, Mass., March 21.  
Annual—New York, N. Y., September 19.  
1872. Semiannual—Boston, Mass., March 16.  
Annual—New York, N. Y., September 17.  
1873. Semiannual—Boston, Mass., March 17.  
Annual—New York, N. Y., September 16.  
1874. Semiannual—Boston, Mass., March 17.  
Annual—Not held, owing to error in date of notices sent out.  
1875. Semiannual—Boston, Mass., March 25.  
Annual—New York, N. Y., September 21.  
1876. Semiannual—Boston, Mass., March 21.  
Annual—New York, N. Y., September 10.  
1877. Semiannual—Boston, Mass., March 20.  
Annual—New York, N. Y., September 18.  
1878. Semiannual—Boston, Mass., March 19.  
Annual—New York, N. Y., September 17.  
1879. Semiannual—Boston, Mass., March 18.  
Annual—New York, N. Y., September 16.  
1880. Semiannual—Boston, Mass., March 16.  
Annual—New York, N. Y., September 1.  
1881. Semiannual—Boston, Mass., March 13.  
Annual—New York, N. Y., September 20.  
1882. Semiannual—Boston, Mass., March 21.  
Annual—New York, N. Y., September 19.  
1883. Semiannual—Boston, Mass., March 20.  
Annual—New York, N. Y., September 18.  
1884. Semiannual—Boston, Mass., March 18.  
Annual—Cincinnati, Ohio, September 16.  
1885. Semiannual—Boston, Mass., March 17.  
Annual—New York, N. Y., December 15.

1886. Semiannual—Boston, Mass. No legal meeting held (no quorum).  
 Annual—New York, N. Y., September 21.
1887. Semiannual—Philadelphia, Pa., March 15.  
 Annual—New York, N. Y., September 20.
1888. Semiannual—Baltimore, Md., March 20.  
 Annual—New York, N. Y., September 18.
1889. Semiannual—Boston, Mass., March 19.  
 Annual—Brooklyn, N. Y., September 17.
1890. Chicago, Ill., September 16 and 17.
1891. Washington, D. C., September 15 and 16.
1892. Boston, Mass., September 20, 21 and 22.
1893. Chicago, Ill., October 17, 18, 19 and 20.
1894. Philadelphia, Pa., September 18, 19 and 20.
1895. Des Moines, Iowa, September 10, 11 and 12.
1896. Buffalo, N. Y., September 1, 2 and 3.
1897. Nashville, Tenn., September 7, 8 and 9.
1898. Omaha, Neb., September 6, 7 and 8.
1899. New York, N. Y., September 5, 6 and 7.
1900. Detroit, Mich., September 4, 5 and 6.
1901. Atlantic City, N. J., September 3, 4 and 5.
1902. Minneapolis, Minn., September 2, 3 and 4.
1903. Ottawa, Canada, September 1, 2, 3 and 4.
1904. St. Louis, Mo., August 16, 17, 18 and 19.
1905. Cleveland, Ohio, August 15, 16, 17 and 18.
1906. New Haven, Conn., August 21, 22, 23 and 24.
1907. Kansas City, Mo., September 10, 11, 12 and 13.
1908. Philadelphia, Pa., September 8, 9, 10 and 11.
1909. Chicago, Ill., September 7, 8, 9 and 10.
1910. San Francisco, Cal., September 6, 7, 8 and 9.
1911. Toronto, Canada, August 21, 22, 23 and 24.
1912. Indianapolis, Indiana, August 27, 28, 29, and 30.
1913. New York, N. Y., September 1, 2, 3, 4 and 5.
1914. No meeting.
1915. Oakland, Cal., August 30, 31, September 1 and 2.



#### PRESIDENTS

- 1863–1864. J. H. STICKNEY, Massachusetts.
- 1864–1865. A. S. COPEMAN, New York.
- 1865–1866. C. M. WOOD, Massachusetts.
- 1866–1867. R. H. CURTIS, New York.
- 1867–1869. R. WOOD, Massachusetts.
- 1869–1871. E. F. THAYER, Massachusetts.
- 1871–1875. A. LARGE, New York.
- 1875–1877. A. LIAUTARD, New York.
- 1877–1879. C. P. LYMAN, Massachusetts.
- 1879–1881. J. L. ROBERTSON, New York.

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1881-1883.	W. BRYDEN, Massachusetts.
1883-1885.	W. B. E. MILLER, New Jersey.
1885-1886.	L. McLEAN, New York.
1886-1887.	A. LIAUTARD, New York.
1887-1889.	R. S. HUIDEKOPER, Pennsylvania.
1889-1890.	C. B. MICHENER, New York.
1890-1892.	R. S. HUIDEKOPER, Pennsylvania.
1892-1893.	W. L. WILLIAMS, Indiana.
1893-1896.	W. HORACE HOSKINS, Pennsylvania.
1896-1897.	F. H. OSGOOD, Massachusetts.
1897-1898.	D. E. SALMON, District of Columbia.
1898-1899.	A. W. CLEMENT, Maryland.
1899-1900.	LEONARD PEARSON, Pennsylvania.
1900-1901.	TAIT BUTLER, Indiana.
1901-1902.	J. F. WINCHESTER, Massachusetts.
1902-1903.	S. STEWART, Missouri.
1903-1904.	R. R. BELL, New York.
1904-1905.	M. E. KNOWLES, Montana.
1905-1906.	W. H. LOWE, New Jersey.
1906-1907.	JAMES LAW, New York.
1907-1908.	W. H. DALRYMPLE, Louisiana.
1908-1909.	J. G. RUTHERFORD, Ontario, Can.
1909-1910.	A. D. MELVIN, District of Columbia.
1910-1911.	G. H. GLOVER, Colorado.
1911-1912.	S. BRENTON, Michigan.
1912-1913.	J. R. MOHLER, District of Columbia.
1913-1915.	C. J. MARSHALL, Pennsylvania.
1915-1916.	R. A. ARCHIBALD, California.



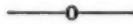
#### SECRETARIES

1863.	A. LIAUTARD, New York.
1864.	R. JENNINGS, New York.
1865-1867.	C. BURDEN, New York.
1867-1869.	J. F. BUDD, New York.
1869-1874.	J. L. ROBERTSON, New York.
1874-1877.	J. D. HOPKINS, New Jersey.
1877-1880.	A. A. HOLCOMBE, New York.
1880-1888.	C. B. MICHENER, New York.
1888-1893.	W. HORACE HOSKINS, Pennsylvania.
1893-1894.	T. J. TURNER, Missouri.
1894-1895.	LEONARD PEARSON, Pennsylvania.
1895-1902.	S. STEWART, Kansas and Missouri.
1902-1906.	JOHN J. REPP, Iowa and Pennsylvania.
1906-1910.	RICHARD P. LYMAN, Connecticut and Missouri.
1910-1913.	C. J. MARSHALL, Pennsylvania.
1913-1915.	NELSON S. MAYO, Illinois.
1915-1916.	C. M. HARING, California.



OFFICERS, 1915-1916

PRESIDENT		
R. A. ARCHIBALD		Oakland, Cal.
FIRST VICE-PRESIDENT		
V. A. MOORE		Ithaca, N. Y.
SECOND VICE-PRESIDENT		
ROBERT W. ELLIS		New York City
THIRD VICE-PRESIDENT		
ADOLPH EICHHORN		Washington, D. C.
FOURTH VICE-PRESIDENT		
C. D. MCGILVRAY		Winnipeg, Man.
FIFTH VICE-PRESIDENT		
GEORGE H. HART		Los Angeles, Cal.
SECRETARY		
C. M. HARING		Berkeley, Cal.
TREASURER		
F. H. SCHNEIDRR		Philadelphia, Pa.
LIBRARIAN		
JAMES N. FROST		Ithaca, N. Y.



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SANITARY SCIENCE AND POLICE

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BOYD, JOHN A., Mason City, Nebr.  
BOYNTON, WILLIAM H., Bureau of Agriculture, Manila, P. I.  
BRACH, M. W., Hales Corners, Wis.  
BRADLEY, CHAS. A., Marion, Ia.

- BRAGINTON, FRED, 1934 N. New Jersey St., Indianapolis, Ind.  
(Mail returned).
- BRAINERD, E., Memphis, Mo.
- BRAISTED, WILLIAM, 235 E. 57th St., New York City. (Mail returned). 1913.
- BRANDENBERG, T. O., Lakota, N. D.
- BRANSON, ROSCOE ARTHUR, R. D. 1., Wichita, Kans.
- BRASHIER, EARL S., 2533 State St., Chicago, Ill.
- BRAZENALL, THOMAS, Bury, Que.
- BRAZIE, F. E., Harlan, Ia.
- BRAY, THOMAS A., Box 364, El Paso, Tex.
- BREED, C. S., 645 West 160th St., New York City.
- BREED, FRANK, Pearl River, N. Y.
- BRENTON, S., 121 West Alexandrine Ave., Detroit, Mich.
- BRENTON, WILLIS L., 224 Alexandrine Ave., W., Detroit, Mich.
- BRETT, GEO. W., 1636 30th St., N. W., Washington, D. C.
- BRIMHALL, S. D., Mayo Clinic Bldg., Rocheste., Minn.
- BRONSON, ELY M., 2946 N. Capitol Ave., Indianapolis, Ind. 1913.
- BROOKBANK, ROSCOE E., Seville Apts., Washington, D. C.
- BROOKS, S. S., Hamilton Ave and 16th St., Brooklyn, N. Y.
- BROSSARD, G. J., Fiefield Row, Ashland, Wis.
- BROTHERIDGE, H. J., 3 16th St., Brooklyn, N. Y. 1913.
- BROUDE, HARRY F., 516 E. State St., Harrisburg, Pa. 1913.
- BROWN, ARTHUR C., 407 P. O. Bldg., San Francisco, Cal. (Mail returned). 1911.
- BROWN, ELDRIDGE N., 316 First Ave., N., Nashville, Tenn. 1910.
- BROWN, F. F., 1336 E. 15th St., Kansas City, Mo.
- BROWN, FRANK H., B. A. I., Danville, Ind.
- BROWN, HERBERT AUSTIN, Box 428, Victoria, B. C. 1910.
- BROWN, LYMAN D., S. Broadway, Hamilton, Mo.
- BROWN, SYLVESTER, 122 S. Union St., Traverse City, Mich.
- BROWNLEE, WM. F., Kirkwood, Ill.
- BROWNING, GEORGE W., Box 309, La Grange, Ga.
- BROWNING, P. H., 66 N. San Pedro St., San Jose, Cal.
- BRUNER, SAMUEL E., Live Stock Sanitary Board, Harrisburg, Pa.
- BRUNS, GEORGE H., 4 Live Stock Record Bldg., Union Stock Yards, Chicago, Ill.
- BRYANS, JOSEPH W. E., Lansford, N. D.
- BRYANT, ALBERT E., Menomonie, Wis.
- BUCHER, CLARENCE S., 1743 Warren Ave., Chicago, Ill. 1914.
- BUCHTEL, JOHN T., Lockhart, Texas. 1914.
- BUCK, JOHN M., B. A. I., Washington, D. C.
- BUCKINGHAM, DAVID E., 2115 14th St., Washington, D. C.
- BUCKLEY, JOHN M., 600 Carlton Ave., Brooklyn, N. Y. 1913.
- BUCKLEY, JOHN S., College Park, Md.
- BUCKLEY, SAMUEL S., College Park, Md. 1914.
- BUENCAMINO, VICTOR, 2229 Azcarraga, Manila, P. I.
- BULLER, JOHN J., 944 4th St., Santa Monica, Cal.



- BULLIVANT, JAMES, Spokane, Wash. 1914.  
BULLOCK, JOHN L., Creedmoor, N. C.  
BUNDY, EDWARD A., 2346 Hudson Ave., Ogden, Utah.  
BURDETT, CYRIL H., 5th and Commercial Sts., Centralia, Kans.  
BURKE, JAMES W., 2012 Canalport Ave., Chicago. (Mail returned) 1912.  
BURKHOLDER, CLINTON E., Chief Lake, Mich. 1911.  
BURKLAND, HERMAN W., 475 N. Boulevard Ave., Atlanta, Ga.  
BURLEY, ARTHUR JAMES, 2519 House St., Cheyenne, Wyo.  
BURNETT, J. F., care N. W. Mounted Police, Regina, Sask. 1910.  
BURNETT, SAMUEL H., 410 University Ave., Ithaca, N. Y.  
BURNHAM, F. E., 728 Ogden Ave., Superior, Wis.  
BURNS, ALBERT H., Hepler, Kans.  
BURNS, JOHN ROBERT, B. A. I., Live Stock Exch. Bldg., Chicago, Ill. 1912.  
BURR, ALEXANDER, Old Court House, Boston, Mass. 1914.  
BURROWS, SAMUEL, 2317 East 89th St., Cleveland, Ohio.  
BURSON, W. H., Athens, Ga.  
BURT, JAMES HENRY, 800 Poyntz Ave., Manhattan, Kans.  
BUSHNELL, FRED F., Harvard, Ill.  
BUSMAN, HERMAN, P. O., Bldg., South Side Station, Omaha, Nebr.  
BUTIN, GEORGE E., 1612 W. 16th St., Kansas City, Mo.  
BUTLER, GEORGE W., 315 Federal Bldg., Indianapolis, Ind.  
BUTTERFIELD, ORLIN F., Libertyville, Ill.  
BUTTERS, J., Box 335, Renville, Minn.  
BUTZ, FRANK R., 3116 Spring Grove Ave., Cincinnati, Ohio.  
BUZZARD, DAVID K., Nappanee, Ind.  
BYERRUM, ROSWELL O., 400 E. 2nd St., Muscatine, Ia.  
BYERS, M. V., Broken Bow, Nebr.  
BYRD, ATVILLE, 2406 E. 9th St., Kansas City, Mo.  
CADY, BERT J., Univ. of California, Berkeley, Cal.  
CADY, HENRY, 23 Washington St., Gloversville, N. Y.  
CADY, P. L., Fremont, Nebr.  
CAHILL, EDWARD A., Lowell, Mass.  
CAHILL, F. M., 9th and Mary Sts., St. Joseph, Mo.  
CALDWELL, FRED W., St. Joseph, Mo. (Mail returned). 1912.  
CALDWELL, J. H., 16 4th St., S. E., Medicine Hat, Alta.  
CALDWELL, WM. A., Edgewood, Cal.  
CALKINS, R. C., Fairbury, Ill.  
CALLICOTE, J. K., 2 East 4th St., Oklahoma City, Okla. 1913.  
CAMBON, FERDINAND J., 303 Perrin Bldg., New Orleans, La.  
CAMERON, ALNE EDWARD, 20 Shaa Road, Acton, London W., Eng.  
CAMPBELL, DELWIN M., 9 So. Clinton St., Chicago, Ill.  
CANT, W. JOHN, Erie, Ill.  
CARLE, EDWARD C., 2517 Lafayette St., St. Joseph, Mo. 1912.  
CARLISLE, T. S., 8425 Germantown Ave., Philadelphia, Pa. 1910.  
CARMACK, RALPH W., Dana, Ind.  
CARNEY, STEPHEN J., 266 Glisan St., Portland, Ore. 1913.

- CARNRITE, JAMES SCHUYLER, Fort Plain, N. Y.  
CARROLL, THOS. B., 106 N. 2nd St., Wilmington, N. C.  
CARROLL, THOS. E., 518 Wall St., Chico, Cal. 1910.  
CARSON, JAMES R., Cicero, Ind. 1914.  
CARSTENSON, L. P., Columbus, Nebr.  
CARTER, ALVA B., Williamsport, Ind.  
CARTER, BARCLAY FRED, 21 W. Fornance St., Norristown, Pa. 1912.  
CARTER, E. B., Austin, Minn.  
CARTER, GEO. H., 219 Tuscola St., Saginaw, Mich.  
CARTER, JOSEPH E., 70 4th St., N., Fargo, N. D. 1914.  
CARTER, ROLLA E., 619 Ohio Ave., Long Beach, Calif.  
CARTER, R. W., Rancocas Stock Farm, Jobstown, N. J.  
CASE, C. H., 50 E. Buchtel Ave., Akron, Ohio.  
CASE, J. C., Peconic, Long Island, N. Y.  
CASE, LEONARD N., Honolulu, T. H. 1913.  
CASEY, CHARLES M., No. 307 Fabius St., Syracuse, N. Y.  
CASPER, GEORGE T., 419 W. Fruit Ave., Albuquerque, N. M.  
CASTOR, THOS., 4914 Frankfort Ave., Philadelphia, Pa.  
CASH, GEO. B., 464 Bathurst St., Toronto, Ont.  
CAVELLE, EDWIN B., Northville, Mich. 1912.  
CAWLEY, A. O., Lewisburg, Pa.  
CECIL, JOSEPH D., Waterloo, Ia.  
CHAMBERLAIN, FRANK W., Box 1022, East Lansing, Mich.  
CHAMBERLIN, LEROY MYLTON, 2503 Hamlin St., N. E., Washington D. C.  
CHANDLER, GEORGE H., Marseilles, Ohio. 1912.  
CHANNING, CHARLES E., Real Estate Trust Bldg, Washington, D.C.  
CHAPMAN, GEORGE W., Webster, S. D.  
CHASE, CHARLES S., Bay Shore, Long Island, N. Y.  
CHENEY, ALONZO HENRY, Polson, Mont. 1914.  
CHENEY, BAILEY E., Health Dept., Corpus Christi, Tex.  
CHENEY, GEORGE LEROY, 265 Park Ave., New Haven, Conn.  
CHERRINGTON, KENNETH G., 152 Webster St., Pawtucket, R. I. 1911.  
CHESLEY, G. E., 215 Huntington St., Rochester, N. H. 1913.  
CHISHOLM, JOSEPH P., Lisbon, N. D.  
CHRISMAN, WM. G., Blacksburg, Va.  
CHRISTIAN, ROBERT V., 1018 Houston St., Manhattan, Kans.  
CHRISTIENSEN, JOSEPH C., Mt. Vernon, S. D. 1910.  
CHRISTIENSEN, OREN A., 4319 Calumet Ave., Chicago, Ill.  
CHRISTIE, NORMAN DOUGLAS, Box 616, Regina, Sask.  
CHRISTOPHER, RALPH E., 423 Cass St., La Crosse, Wis.  
CLANCY, J. B., Amberston Farm, Edgemont St'n., E. St. Louis, Ill.  
CLARK, B. L., Monticello, Wis.  
CLARK, CLARENCE W., Rice Lake, Wis.  
CLARK, CURTIS ALFRED, College Corner, Ohio.  
CLARK, DAVID BERT, Purdue Univ., Lafayette, Ind.  
CLARK, HENRY D., 69 High St., Fitchburg, Mass.  
CLARK, REES, Elberton, Ga. 1912.

- CLARK, ROY RALPH, Hampton Institute, Hampton, Va.  
CLARK, W. G., Box 196, Marinette, Wis.  
CLARK, BURNETT L., Monticello, Wis.  
CLAWSON, C. A., 2445 Talbott Ave., Indianapolis, Ind.  
CLAYTON, CHAS. E., 207 West 55th St., New York.  
CLEMONS, W. E., Granville, Ohio.  
CLERE, RALPH, W., E. Syracuse, New York.  
CLEVELAND, EDGAR CHARLES, JR., Cattaraugus, N. Y.  
CLEVELAND, WALTER J., Havelock, Ia.  
CLIFFE, G. W., 119 West Johnson St., Upper Sandusky, Ohio.  
CLINE, CLIFFORD, M., Box 298, Maryville, Mo.  
CLINE, GORDON L., Western, Nebr. 1911.  
CLINE, J. D., Clarion, Ia.  
CLOSSON, GARDNER W., 131 Philadelphia St., Anaheim, Cal.  
CLOSE, F. W., Lewiston, Ida.  
COCHRAN, D. W., 19 Vestry St., New York City.  
COCHRANE, ROBERT E., 450 Greenbush St., Milwaukee, Wis.  
COFFEEN, ROBERT J., 225 North 2nd St., Stillwater, Minn.  
COLE, ALONZO B., Montrose, Pa. 1912.  
COLLETT, HOWARD BARKER, care of P. Burns & Co., Calgary, Alta.  
COLLINS, FRANK, 320 N. 6th St., Monroe, La.  
COLLINS, FRED W., Madison, Nebr.  
COLLINS, GEORGE J., West Point, Nebr.  
COLLINS, LEONARD, Stanton, Nebr.  
COLLINS, ROBERT E., 256 Monroe St., Memphis, Tenn.  
COLTON, CHAS. L., 99 Ann St., Hartford, Conn.  
COMMINS, FREDERICK E., 119 Cortland Ave., San Francisco, Cal.  
COMSTOCK, DAVID B., 175 Jay St., Albany, N. Y.  
CONNOWAY, J. W., Univ. of Mo., Columbia, Mo.  
CONNELL, CLARE V., N. 2nd St., Decatur, Ind.  
CONRAD, BURTON W., Sabetha, Kans. 1914.  
COOK, F. G., 220 S. Main St., Paris, Texas.  
COOK, J. W., Brownsville, Ore.  
COOK, L. P., 3116 Spring Grove Ave., Cincinnati, Ohio.  
COOLEY, A. S., State Veterinarian, Columbus, Ohio.  
COOPER, EDWARD, R. F. D., No. 3, Sunman, Ind.  
COOPER, J. M., 711 Plum St., Cincinnati, Ohio.  
COOVER, W. E., Indianapolis, Ind. (Mail returned). 1912.  
COPITHORN, HARRY K., Chelsea, Mass.  
CORBIN, CECIL J., Pawling, N. Y.  
CORBIN, E. A., Tucumcari, N. M. 1912.  
CORCORAN, MICHAEL, Box 142, Augusta Kans.  
CORN, SAMUEL, 5518 Market St., Philadelphia, Pa. 1913.  
CORNMAN, ERNEST L., Marietta, Pa.  
CORWIN, GEORGE E., Canaan, Conn.  
CORWIN, WILLIS T., Pine Island, Minn.  
COSFORD, SAMUEL E., Box 322, Beatrice, Nebr.  
COTTON, CHAS. E., 615 4th Ave., Minneapolis, Minn.

- COTTON, WM. E., 3242 38th St. N. W., Washington, D. C.  
COTTRILL, R. B., Sand Fork, W. Va.  
COURTRIGHT, JOHN M., Lathrop, Mo.  
COUTURE, J. A., 49 Garden St., Quebec, Can.  
COWGILL, DANIEL L., Rio, Wis.  
COX, ABRAHAM G., Carlisle, Ind.  
COX, HARRY B., 1516 Snyder Ave., Philadelphia, Pa. 1914.  
COX, WALTER P., 7 N. Carey St., Baltimore, Md.  
COX, WALTER R., Clio, Mich.  
COXE, S. A., 140 9th St., Brandon, Man., Can.  
COZIER, CARL, 320 Prospect St., Bellingham, Wash.  
CRAIG, ROBERT A., 621 Owen St., Lafayette, Ind.  
CRAIG, THOMAS F., Hampden, N. D.  
CRAIG, W. B., 216 Meridian St., Indianapolis, Ind. 1914.  
CRANE, ADELBERT G., 1639 Wabash Avenue., Chicago, Ill.  
CRAM, V. E., Calexico, Cal. 1912.  
CRANWELL, JOHN JAMES, 642 West Pike St., Clarksburg, W. Va.  
CRAWFORD, HARRY C., 38 Lexington Ave., New York, N. Y.  
CRAWFORD, J. H., Harvard, Ill. 1913.  
CRAWFORD, JAMES E., Far Rockaway, Long Island, N. Y.  
CREAMER, J. M., 5th and Taylor St., Portland, Ore.  
CREECH, G. TINSLEY, 4193 S. Halstead St., Chicago, Ill.  
CREWE, W. F., Bismarek, N. D.  
CRISLER, OTTO S., 209 E. 4th St., Newport, Ky.  
CROCKER, WALTER JAMES, 39th & Woodland Ave., Philadelphia, Pa.  
CULVER, FREDERICK W., Longmont, Colo.  
CUMMING, DAVID, Port Huron, Mich. (Mail Returned). 1911.  
CUNNINGHAM, A. E., 3826 Carnegie St., S. E., Cleveland, O.  
CUNNINGHAM, ELMER E., 106 Indiana Ave., Valparaiso, Ind. 1913.  
CURRAN, MATTHEW JOHN, 39 N. 6th St., New Bedford, Mass.  
CURRY, J. M., 995 Main St., Hartford, Conn. 1911.  
CURTICE, COOPER, U. S. Sheep Farm, R. R. No. 2, Vienna, Va.  
CURTIS, WILBUR A., Plainview, Tex.  
CUSACK, FRANK L., Carrington, N. D.  
CUSTIS, HOWARD H., La Jara, Colo.  
DALRYMPLE, W. H., Baton Rouge, La.  
DAMMAN, ARTHUR J., 749 12th Ave. E., Vancouver, B. C., Can.  
DANFORTH, ARTHUR L., 103 Orchard St., Watertown, N. Y.  
DANIELSON, LEOPOLD A., Madera, Cal.  
DARRAH, GEORGE D., 648 Hall St., Manchester, N. H.  
DARROW, JOHN HENRY, JR., 78 N. Hamilton St., Poughkeepsie, N. Y.  
DAUBER, CHAS. C., 107 N. Clay St., Sturgis, Mich.  
DAUBIGNY, F. T., 12 Rue St. Denis, Montreal, Que. 1914.  
DAUGHERTY, T. E., Danville, Ind. 1913.  
DAUTH, ALBERT, Coteau, DuLac, Que. 1910.  
DAVENPORT, MILES L., Fergus Falls, Minn.  
DAVIDSON, GEORGE H., Rugby, N. D.  
DAVIDSON, WILLIAM A., Exchange Bldg., S. St. Joseph, Mo.



- DAVIS, BENJAMIN F., Box 509, Douglas, Wyo.  
DAVIS, HARRY EMERSON, Arlington, S. D.  
DAVIS, J. E., Hume, Ill. 1912.  
DAVIS, WILLIAM L., B. A. I., S. Omaha, Nebr. (Mail Ret.) 1914.  
DAVISON, ELWIN T., Athenia, N. J.  
DAVITT, M. H., 525 N. Main St., Palmer, Mass.  
DAWDY, CLARENCE A., Brawley, Cal. 1912.  
DAY, FRANK J., 50 Rhode Island Ave., N. W., Washington, D. C.  
DAY, L. ENOS., 4193 S. Halstead St., Chicago, Ill.  
DEADMAN, CHARLES A., 311 E. Main St., Madison, Wis.  
DECKER, E. J., 16 William St., Far Rockaway, Long Island, N. Y.  
DEILING, N. J., Dallas Center, Ia.  
DELL, JESSE APPLIN, 16th and Pacific Aves., Los Angeles, Cal.  
DELLERT, R. V., 568 Banning St., Winnipeg, Can.  
DEMING, CHARLES W., Great Falls, Mont., Box 1287. 1912.  
DEMING, S. A., Ida Grove, Ia.  
DEMOTT, LEE, Petersburg, Ind.  
DENISON, WILLIAM K., Bemidji, Minn.  
DERONDE, JOHN D., 48 East 89th St., New York City.  
DESERPA, JOHN A., Box 614, Oxnard, Cal.  
DESMOND, J., 77 Currie St., Adelaide, S. Australia (Deceased).  
DEUBLER, ERNEST C., Wayne, Pa.  
DEUBLER, EZRA S., Narberth, Pa.  
DEVEREAUX, JOHN L., 20 Brook St., Waterbury, Conn.  
DEVINE, JOHN F., Goshen, N. Y.  
DICK, GEORGE A., Kane, Pa.  
DICKY, GEO. W., 319 N. Weber St., Colo. Springs, Colo.  
DICKSON, JOHN, 444 Federal Bldg., Denver, Colo.  
DILL, BENNIE G., Charleston, S. C. (Deceased Sept. 17, 1915).  
DILLAHUNT, PETER A., Springfield, Ohio, R. F. D. No. 7.  
DILLON, L. RAY, 107 N. Grand Ave., Pueblo, Colo.  
DIMOCK, WM. WALLACE, Iowa State College, Ames, Ia.  
DINGLEY, ERNEST C., 5335 DeLancey St., Philadelphia, Pa.  
DINWIDDIE, R. R., Ft. Smith, Ark. 1912.  
DINWOODIE, JOHN T. E., Brookings, S. D.  
DITEWIG, GEORGE, B. A. I., Washington, D. C.  
DITTO, JOHN K., Pleasureville, Ky.  
DIVINE, JOHN P., Ballston, Va. 1914.  
DIXON, C. PRICE, Old Fire House, Charlottesville, Va. 1914.  
DIXON, H. L., Box 616, Regina, Sask, Can.  
DOBBINS, QUINCY C., Bedford, Ind.  
DOBSON, CHARLES C., Muncie, Ind.  
DODGE, GEORGE A., Northwood, Ia. 1911:  
DODGE, WILLIAM H., Leominster, Mass.  
DOLAN, FRANCIS F., Willow City, N. D.  
DONALD, JAMES S., 213 S. Catherine St., Bay City, Mich.  
DONNELLY, GEORGE J., 833 Telegraph Ave., Oakland, Cal.  
DONOHUE, ROBERT JOHN, 506 Alaska Bldg., Seattle, Wash.

- DORAN, JOHN THOMAS, Beatrice, Nebr.  
DORIAN, FRANK P., 35 Franklin St., Yonkers, N. Y.  
DOUGLASS, FRANK J., 1235 St. Andrew, New Orleans, La.  
DOYLE, SIMON A., 1764 Mission St., San Francisco, Cal. 1913.  
DRAKE, EDW. J., Toledo, Wash. (Mail returned). 1914.  
DRAKE, M. W., 1308 Morris, Philadelphia, Pa.  
DRAPER, JAMES POWELL, Lowell, Mich.  
DREHER, WM. HENRY, Box 143, Oregon, Wis.  
DREPPARD, SAMUEL G., Flora, Ill.  
DREXLER, JOSEPH L., Thibodaux, La.  
DRURY, JAMES, 218 Michigan St., Ypsilanti, Mich.  
DUFRESNE, A. A., Longueuil, Quebec, Can.  
DU FRENE, ALFRED J., Glendive, Mont.  
DU JARDIN, GUILLAUME, 270 Craig St., E., Montreal, Que. 1914.  
DUNLEAVY, M. J., 1324 Acoma St., Denver, Colo.  
DUNN, RALPH C., College Station, Tex.  
DUNPHY, G. W., East Lansing, Mich.  
DUSTAN, HENRY W., 25 DeHart St., Morristown, N. J.  
DYKSTRA, RALPH R., 714 Houston St., Manhattan, Kan.  
DYSON, ORION E., State Veterinarian, Springfield, Ill.  
EAGAN, PAUL HERMAN, 817 N. 7th St., East St. Louis, Ill.  
EAGLE, ALEX. F., 725 San Jose Ave., San Francisco, Cal.  
EAGLE, JOHN G., 3924 Morrel Ave., Kansas City, Mo.  
EAGLE, RICHARD F., 1309 N. Ellison St., Oklahoma City, Okla.  
EAGLE, WM. W., 3rd and Central Ave., Kansas City, Kan.  
EASTMAN, O. R., Gadsden, Ala.  
EBBITT, RICHARD, Naper Arms, Old Castle, C. Meath, Ireland.  
ECKERT, HENRY F., Markesan, Wis.  
EDELIN, ALLEN ALFRED, 1602 Lagonda St., Fort Worth, Tex.  
EDGINGTON, BRUCE H., Reynoldsburg, Ohio.  
EDMONDS, ELMER V., 603 Lincoln Ave., Mt. Vernon, Wash.  
EDMONDS, ARTHUR L., Franklin, N. H.  
EDWARDS, FRANK, Bureau of Agriculture, Manila, P. I.  
EDWARDS, IRA W., Box 82, Stuart, Iowa.  
EDWARDS, W. R., Box 216, Vicksburg, Miss.  
EGAN, JOHN MILTON, 1155 Golden Gate Ave., San Francisco, Cal.  
EGAN, PETER J., 1155 Golden Gate Ave., San Francisco, Cal.  
EGAN, WILLIAM F., 1155 Golden Gate Ave., San Francisco, Cal.  
EGBERT, ARCH, 411 E. 4th St., Logan, Utah. 1911.  
EICHELBERGER, A. MARTIN, Box 488, Spring and Milan Sts.,  
Shreveport, La.  
EICHHORN, ADOLPH, B. A. I., Washington, D. C.  
EISEMAN, FRANK T., 222 East Main St., Louisville, Ky. 1913.  
EISENHOWER, ELMER C., Gypsum, Kans.  
EISENHOWER, JAMES M., Schell City, Mo.  
EISENLOHR, HERMAN M., Larimore, N. D.  
ELERY, WILTON, Audubon, Ia.  
ELIASON, OSCAR H., State Veterinarian, Madison, Wis.

- ELKIN, ALBERT F., R. F. D. No. 1, Smicksburg, Pa.  
ELKIN, JOSEPH HOMER, Smicksburg, Pa.  
ELLENBERGER, W. P., 1359 Parkwood Place., Washington, D. C.  
ELLIOTT, ADAM F., Milton, N. D.  
ELLIOTT, CHAS. M., Seward, Nebr.  
ELLIOTT, CLARENCE L., 5434 S. 3rd St., So. St. Joseph, Mo.  
ELLIOTT, EDWARD W., Park River, N. D.  
ELLIOTT, HAROLD B., P. O. Box 167, Hilo, Hawaii.  
ELLIS, ROBERT W., 509 W. 152nd St., New York City.  
ELLIS, PERCY L., Box 272, Merrill, Ia.  
ELSEY, MARK A., Marion, Ohio.  
ELWELL, FRED N., National Stock Yards, St. Claire Co., Ill.  
ELZINGA, MARTIN E., 349 La Grave Ave., Grand Rapids, Mich.  
EMBREE, WARREN J., Aberdeen, S. D. (Mail returned). 1911.  
ENGEL, JOHN H., 1311 Harford Ave., Baltimore, Md. 1912.  
ENGLE, FRANK PHILLIP, Ft. Worth, Tex. (Mail returned). 1913.  
ERNST, JOHN, JR., 125 East 4th South St., Salt Lake City, Utah.  
ESSEX, JOHN J., Chevy Chase, Md.  
ESTEY, CYREW B., Box 287, St. Cloud, Minn.  
ETIENNE, ALBERT A., 67 Drummond St., Montreal, Can.  
ETTLING, CHRISTIAN C., 910 3rd St., E. Las Vegas, N. M. 1911.  
EVANS, CHRISTMAN E., 719 Wisconsin St., Racine, Wis.  
EVANS, CALVIN S., 323 Exchange Bldg., Sioux City, Ia. 1911.  
EVERETT, A. T., 24th and North Sts., So. Omaha, Nebr.  
EWALT, W. AUSTIN, 22 Grand Ave., Mt. Clemens, Mich.  
EXLINE, JAMES C., 311 Live Stock Ex. Bldg., Denver, Colo.  
FABIAN, ARTHUR H., 421 Walworth St., Lake Geneva, Wis.  
FAIR, J. D., Millersburg, Ohio.  
FAKE, CHARLES THOMPSON, Granville, N. Y.  
FALCONER, THOMAS, Box 303, Alexandria, Minn.  
FALLON, EDWARD J., 3372 A 16th St., San Francisco, Cal.  
FARLEY, A. J., 873 Towne Ave., Pomona, Cal.  
FARMER, ALBION C., Berlin, N. H.  
FARMER, THOMAS, Grand Blanc, Mich.  
FARRINGTON, A. M., 1436 Chapin St., Washington, D. C.  
FAUST, OTTO, 209 Union Ave., Poughkeepsie, N. Y.  
FAVILLE, G. C., North Emporia, Va. 1913.  
FEELEY, ROBERT O., Clemson College, S. C.  
FEIST, ARNOLD ANDREW, 156 Aurora Ave., St. Paul, Minn.  
FERGUSON, CHAS. W., Auburn, Ala.  
FERGUSON, THOMAS H., 421 Broad St., Lake Geneva, Wis.  
FERGUSON, W. P., 316 Second St., Grenada, Miss.  
FERNEYHOUGH, JAMES G., Oak St., Burkville, Va. (Mail returned). 1914.  
FERNEYHOUGH, R. E., Warrenton, Va., Lee St.  
FERNSLER, FRANK U., 32 South 7th St., Lebanon, Pa.  
FERRAND, WILLIAM S., Gilmore City, Ia.  
FERRO, R. B., Gordon Hotel, Lafayette, La.

- FETHEROLF, GEO. R., Reading, Pa. 1914.  
FINDLAY, ALEXANDER, 11 Main St., Camden, N. Y. (Mail returned). 1912.  
FINKLE, RAY C., Seymour, Wis.  
FINLEY, LESTER C., Lapel, Ind.  
FISCHER, CARL F., Garden City, Mo.  
FISCHER, HERMAN C., Bellair, Mich. 1912.  
FISCHER, PAUL, Reynoldsburg, Ohio.  
FISH, PIERRE A., N. Y. State Vet. Col., Ithaca, N. Y.  
FISHER, ADAM, 9 West 4th St., Charlotte, N. C.  
FISHER, CARL W., 420 A St., San Mateo, Cal.  
FISHER, D. Grandin, N. Dak.  
FISHER, LAWRENCE W., Bureau of Agriculture, Manila, P. I. 1912  
FISK, ALEXANDER G., 815 Willow St., Trinidad, Colo.  
FITCH, CLIFFORD PENNY, 107 Brandon Place, Ithaca, N. Y.  
FITCH, EARL W., Arcade, N. Y.  
FITZPATRICK, DENNIS B., 3225 Woodland Ave., Philadelphia, Pa.  
FLAHERTY, JAMES J., 127 Meadow St., New Haven, Conn. 1913.  
FLANARY, W. F., St. Charles, Minn.  
FLEMING, CHARLES I., 20 West 5th St., Terre Haute, Ind. 1912.  
FLEMING, W. B., 12 Washington St., Montgomery, Ala. 1911.  
FLEMING, W. R., 25 Live Stock Exchange, Buffalo, N. Y. 1913.  
FLOCKEN, CHARLES F., Experiment Station, St. Anthony Park, Minn.  
FLOWER, E. PEGRAM, Box 24, Baton Rouge, La.  
FLOWERS, ROYAL G., 3rd and Main Sts., Ft. Worth, Tex.  
FOGLE, CHARLES W., Leipsic, Ohio.  
FOLSE, CHARLES D., 1336 E. 15th St., Kansas City, Mo.  
FOLSOM, EDWARD GRAHAM, Watson Bldg., Fairmount, W. Va.  
FOOS, ARTHUR C., 126 N. Laurel St., Hazelton, Pa.  
FORBES, S. D., 301 Cameron St., Alexandria, Va.  
FORGE, LOUIS A., 561 Washington St., Burlington, Wis.  
FORMAD, ROBERT J., B. A. I., Washington, D. C.  
FOSBINDER, HARRY R., 1622 Cosmo St., Hollywood, Cal.  
FOSTER, ALLEN A., 3813 Ross Ave., Dallas, Tex.  
FOSTER, FRED, 2nd Field Artillery, Vancouver Barracks, Wash. 1910.  
FOSTER, J. D., 126 N. State St., Newtown, Pa.  
FOSTER, JOAB P., Box 27, Bangor, Me.  
FOSTER, ROBERT J., 9th U. S. Cavalry, Manila, P. I.  
FOSTER, SAMUEL B., 927 Union Ave., North, Portland, Ore.  
FOSTER, THOMAS J., Monticello, Ill.  
FOWLER, W. J. R., 52 Pacific Ave., Toronto, Ontario, Can.  
FOX, DAVID F., Room 423 First Nat'l Bank Bldg., Oakland, Cal.  
FRANCUS, M., College Station, Tex.  
FRANCOISE, WM. I., 605 East Ave., Kalamazoo, Mich. 1914.  
FRANK, JOHN WILLIAMSON, Box 291, Nelson, B. C.  
FRANZMAN, PETER A., 5966 Lowe Ave., Chicago, Ill.



- FRASER, THOMAS, 316 N. Henry St., Richmond, Va.  
FRASER, WALTER, 13th U. S. Cavalry, Marfa, Tex.  
FRAZIER, CHAS., 5052 Cottage Grove Ave., Chicago, Ill.  
FREDERICK, CHARLES B., 412 N. Walnut St., N. E., Canton, Ohio.  
1914.  
FREDERICK, HARRY, Box 584, Suffern, N. Y.  
FREDERICK, HYRAM, J., College Hill, Logan, Utah.  
FREDERICKS, WM. J., Franklin Ave., Delawanna, N. J. 1912.  
FREED, B. M., 12 South Dock St., Sharon, Pa.  
FREEMAN, F. E., Buhl, Ida.  
FRENCH, ALEX. W., Cheyenne, Wyo.  
FRENCH, WM. HAROLD, Redfield, S. D. 1913.  
FRESE, GEORGE L., 309 11th St., Toledo, Ohio.  
FREY, CHARLES T., Box 106, River Point, R. I.  
FRIEDHEIM, LOUIS, Box 103, Rock Hill, S. C.  
FRIDIRICI, ULYSES G., 204 Pine St., Tamaqua, Pa.  
FROST, JAMES N., 919 East State St., Ithaca, N. Y.  
FROST, ROY C., 1513 U. St., N. W., Washington, D. C.  
FROTHINGHAM, LANGDON, 335 Bay State Road, Boston, Mass.  
FULLER, CLAUDE E., Beach, N. D.  
FULLER, GEORGE S., 170 Common St., Lawrence, Mass.  
FULLER, GEORGE S., 1715 N. 12th St., Philadelphia, Pa.  
FULLER, JOHN RUSSEL, 101 W. Commercial St., Weiser, Ida.  
FULLINGTON, HARRY C., 6047 Greenwood Ave., Seattle, Wash.  
1913.  
FULSTOW, HARRY, Norwalk, Ohio.  
FUNKHOUSER, GEORGE M., 410 Main St., Lafayette, Ind.  
GAIN, J. H., State Farm, Lincoln, Nebr.  
GAINES, CHAS. H., Chilhowee, Mo.  
GALL, WM., Mattawan, N. J.  
GALLAGHER, BERNARD A., B. A. I., Washington, D. C.  
GALLAGHER, JOHN J., Western Ave., Lovelock, Nev.  
GALLIVAN, MICHAEL V., P. O. Box 567, Lethbridge, Alberta.  
GALLOWAY, PETER F., 318 N. Henry St., Richmond, Va.  
GAMBLE, HENRY S., 1329 Gallatin N. W., Washington, D. C.  
GAMRATH, CARL L., 52 E. Washington St., Fairfield, Ia.  
GANNETT, RAY WILLARD, 186 Sterling Ave., Brooklyn, N. Y.  
GARDNER, CHENNIE A., Morris, N. Y.  
GARSDIE, PETER, Bourbon, Ind.  
GATES, WM. L., Clarksdale, Miss.  
GAY, CARL W., Swarthmore, Pa.  
GEARHART, FRANK C., Bureau of Agriculture, Manila, P. I., Box  
1362.  
GEMMILL, A. D., 220 E. Livingston St., Celina, Ohio.  
GEORGE, HARRISON H., 235 Federal Bldg., Cleveland, Ohio.  
GEORGE, HERBERT H. S., Kamloops, B. C.  
GERLACH, G. H., Morenci, Mich. 1913.  
GIBSON, A., 1617 2nd St., Birmingham, Ala.

- GIBSON, HOWARD RENWICK, Algona, Ia.  
GIBSON, G. D., Adrian, Mich.  
GIBSON, JAMES IRVINE, State House, Des Moines, Ia.  
GIFFEE, JOE W., (1816 Missouri Ave.) South Side Station, Omaha, Nebr.  
GILCHRIST, WM. T., 410 Church St., Norfolk, Va.  
GILES, WALTER M., Franklin, Tenn.  
GILL, H., 337 E. 57th St., New York City.  
GILL, JOSEPH C., 2nd and Main Sts., Clarksville, Tenn. 1910.  
GILLESPIE, JOHN F., 446 Hudson Ave., Brooklyn, N. Y.  
GILLIE, PETER T., 22 E. First St., Mansfield, Ohio.  
GILLILAND, S. H., Marietta, Pa.  
GILTNER, L. T., Box 3, Bismarck, N. D.  
GILTNER, WARD, East Lansing, Mich.  
GILYARD, ARTHUR T., 74 Phoenix Ave., Waterbury, Conn.  
GIMPER, W. S., Live Stock Sanitary Board, Harrisburg, Pa.  
(Mail returned). 1912.  
GLEASON, MATTHEW E., Fowlerton, Tex.  
GLEDENNING, C. G., 115 W. Washington St., Clinton, Ill.  
GLENNON, HENRY J., 148 Second St., Newark, N. J.  
GLENNON, JAMES T., 27 Clay St., Newark, N. J.  
GLOVER, ALBERT D., Newark, Mo.  
GLOVER, GEORGE H., Fort Collins, Colo.  
GLYNN, LAWRENCE L., 444 Federal Bldg., Denver, Colo.  
GOHN, HORACE M., St. Johns, Mich.  
GOLDING, CYRIL, Tulare St., Dinuba, Cal.  
GOODWIN, JAS. ARTHUR, Lock Box 584, New Iberia, La.  
GOODWIN, PERCY W., Wrights, Cal.  
GORDON, GEORGE, Hanford, Cal.  
GORDON, GEORGE, Gibson City, Ill.  
GORDON, WALDRON M., 1513 6th Ave., Sioux City, Ia. 1912.  
GORDON, WILLIAM D., 268 West Santa Clara St., San Jose, Cal.  
GORE, TRUMAN E., 111 W. Pike St., Clarksburg, W. Va.  
GORSUCH, DICKINSON, Glencoe, Md.  
GOSS, LEONARD W., Agricultural College, Manhattan, Kans.  
GOULD, J. H., 2nd F. A., Manila, P. I.  
GOULD, J. N., Worthington, Minn.  
GOW, RONALD M., State Veterinarian, Old State House, Little Rock, Ark.  
GRADY, WILLIAM J., Lankin, N. D. 1914.  
GRAF, JOHN, 2nd Ave., West, Cresco, Ia.  
GRAFF, CARL L. P., Bisbee, N. D.  
GRAHAM, G. G., 1228 Main St., Kansas City, Mo.  
GRAHAM, JAMES, 115 Queen St., Germantown, Philadelphia, Pa.  
GRAHAM, JOHN J., West Queen Lane, Germantown, Pa. 1914.  
GRAHAM, LEROY B., 369 S. 18th St., Cedar Rapids, Ia.  
GRAHAM, OSWALD HOOD, Clinton, N. C.  
GRAHAM, RALPH, National Stock Yards, Ill. 1911.

- GRAHAM, ROBERT, Experiment Station, Lexington, Ky.  
GRAPP, GUSTAV H., Port Deposit, Md.  
GRANGE, E. A. A., Ontario Vet. College, Toronto, Ont., Can.  
GRAU, JEPPE ANDREW, Tecumseh, Neb.  
GRAVES, FRED W., New Richmond, Ind. 1912.  
GRAVES, HENRY T., E. 201 15th St., Olympia, Wash.  
GRAY, FRED SUMNER, Box 561, Miles City, Mont.  
GRAYBILL, GUY M., Milford Square, Pa.  
GRAYBILL, HARRY W., 1021 Daisy Ave., Long Beach, Cal.  
GREEDER, HERMAN, Elkhorn, Wis.  
GREEN, L. KENNETH, 37 Knight St., Auburn, Maine.  
GREER, JOHN, Saranac Lake, N. Y.  
GREESON, J. O., Kokomo, Ind.  
GRIFFITH, J. W., Cedar Rapids, Ia.  
GRIFFITH, ROSCOE C., Jamestown, Ohio.  
GROFF, BENJAMIN WARREN, 20 North St., Massillon, Ohio.  
GROGAN, JOSEPH P., 909 Ashland Ave., Baltimore, Md.  
GROSS, JNO. L., Bureau of Agriculture, Manila, P. I.  
GROSS, R. C., Elizabethtown, Pa.  
GROSSMAN, JAMES D., 117 Ash Ave., Ames, Iowa.  
GROVE, JNO. S., 310 Federal Bldg., Oklahoma, Okla.  
GROVER, ARTHUR L., 65 9th Ave., New York City. 1914.  
GROVES, JOHN W., 40 York St., Hamilton, Ont.  
GRUBB, CHAUNCEY M., Box 202, Rockville, Md.  
GRUBER, JOHN T., 316 N. Main St., Marion, Ohio.  
GRUENEWALD, GEO. J., B. A. I., Federal Bldg., Cleveland, Ohio.  
GRUNER, WALTER H., 225 Mary St., Evansville, Ind. 1912.  
GRUTZMAN, WALTER R., Ft. Bliss, Tex. 8th Cavalry.  
GUARD, WILLARD F., Veterinary College, Ames, Ia.  
GULDAGER, FRED HOWARD, 304 Ridge St., Sacramento, Cal.. 1913.  
GUYSELMAN, P. C., Monte Vista, Colo.  
GYSEL, ROBERT, 9333 Escanaba St., Chicago, Ill.  
HADLEY, FREDERICK B., Univ. of Wis., Madison, Wis.  
HADWEN, SEYMOUR, Agassiz, B. C.  
HAFFER, JOHN W., 49 Pearl St., Paterson, N. J.  
HAINES, W. ALBERTSON, Bristol, Pa.  
HALL, ADRIAN V., Oxford, Pa. 1910.  
HALL, ORLAN, Health of Animals Branch, Ottawa, Ont., Can.  
HALLIDAY, ROBERT J., 21 West 32nd St., Bayonne, N. J.  
HALLMAN, ELAM T., 383 Sunset Lane, East Lansing, Mich.  
HALLORAN, JOHN L., Broad St., Stapleton, Staten Island, N. Y.  
HALLQUIST, RALPH A., Box 182, Oslo, Minn.  
HALSTED, WILLIAM E., 19 Judson St., Binghamton, N. Y.  
HALTON, JOHN H., 123 G. St., Salt Lake City, Utah.  
HALVERSON, HAROLD M., Box 354, Yankton, S. D.  
HAMBLET, C. A., 495 Varnum Ave., Lowell, Mass.  
HAMILTON, GEORGE W., 604 N. 10th St., E. St. Louis, Ill.  
HAMILTON, HERBERT B., 79 Hillman St., New Bedford, Mass.

- HAMILTON, HOWARD M., Paris, Ky.  
HAMILTON, M., 364 Main St., Delhi, N. Y.  
HAMILTON, ROBERT, 1420 Fort St., Victoria, B. C. 1913.  
HAMILTON, WM. C., Union Stock Yards, Chicago, Ill. (Mail returned). 1912.  
HAMMOND, HARRY J., Box 338, Sacramento, Cal.  
HANAWALT, DAVID C., Laurel, Miss. 1912.  
HANDLEY, JOHN ISAAC, Box 8, West Raleigh, N. C.  
HANDLEY, JNO. M., Woodworth, Wis.  
HANEY, W. F., 1518 Ninth St., Modesto, Cal.  
HANNA, ROBERT LEE., Brookville, Ind. 1912.  
HANSEN, HANS P., 216 E. Mill St., Austin, Minn.  
HANSEN, JAMES W. G., 511 N. Lafayette St., Greenville, Mich.  
HANSHAW, E., 125 Carlton Ave., Brooklyn, N. Y.  
HANSON, H. D., Darien, Conn.  
HANVEY, GEORGE A., 5th Cavalry, Harlingen, Tex.  
HARDENBERG, JAMES BALL, 39th and Woodland Ave., Philadelphia, Pa.  
HARDMAN, RUSSELL T., Kyger, W. Va.  
HARGRAVE, J. C., Dominion Vet. Inst., Medicine Hat, Alta.  
HARING, C. M., Div. of Vet. Science, Univ. of Cal., Berkeley, Cal.  
HARKINS, MALCOLM J., care H. K. Mulford Co., Glenolden, Pa.  
HARMS, HERBERT F., Pearl River, Rockland Co., N. Y. 1912.  
HARRIS, THOS. B., 22nd Divisional Train, B. E. F., France.  
HARRINGTON, E. T., 873 Broadway, S. Boston, Mass. 1911.  
HARRIS, A. W., 78 Fourth Ave., Ottawa, Can.  
HARRIS, E. D., Casselton, N. D.  
HARRIS, J. G., Duluth, Minn. 1912.  
HARRISON, JAMES V. S., 120 South Pitcher St., Kalamazoo, Mich.  
HARRISON, W. F., 329 Broad St., Bloomfield, N. J.  
HARRY, CHARLES EDWARD, Anita, Ia.  
HARSH, FRANCIS A., 326 Murray Ave., Minerva, Ohio.  
HART, CHARLES HENRY, Hankinson, N. D.  
HART, GEORGE H., Health Office, City Hall, Los Angeles, Cal.  
HART, JOHN P., Winchester, Ind. 1913.  
HART, WM. J., Wetmore, Kans.  
HARTHILL, ALEXANDER, 707 Green St., Louisville, Ky. 1914.  
HARTMAN, THOS. T., 1027 Ann St., Kansas City, Kan. 1910.  
HARTMAN, WM. J., State College of Agriculture, Bozeman, Mont.  
HASSELBALCH, A. E., St. Edward, Nebr. 1914.  
HASSLOCH, AUGUST, 400 West 50th St., New York City, N. Y.  
HATTERSCHEID, CHAS. A., Aberdeen, S. D. 1910.  
HAWKE, WALTER L., Lethbridge, Alta.  
HAWKE, WILLIAM RICHARD, Medicine Hat, Alta. 1912.  
HAWKINS, JOSEPH, 184 Stanton Ave., Detroit, Mich. 1913.  
HAWORTH, CHARLES C., Donnellson, Ill.  
HAXBY, J. W., 6th St., and 3rd Ave., Clarinda, Ia.  
HAY, LEOPOLD, 5th and 1st Ave., Fairbault, Minn.



- HAYDEN, CHARLES ERNEST, 108 Irving Place, Ithaca, N. Y.  
HAYES, FRED M., Univ. Farm, Davis, Cal.  
HAYES, JESSE, Alexandria, Va.  
HAYES, JOHN J., 7 E. 42nd St., New York City.  
HAZEL, GEORGE A., 113 East 83rd St., New York City. 1913.  
HAZLET, SAMUEL K., Oelwein, Ia.  
HAZLEWOOD, ROBERT V., Bessemer, Ala.  
HEACOCK, CLYDE C., Carlsbad, N. M. 1913.  
HEAD, CHARLES, Regina, Sask., Can. 1911.  
HEALEY, THOM. W., 461 North 2nd St., San Jose, Cal.  
HEATH, WARREN E., Columbus, Mont.  
HEATON, JOHN B., Indianapolis, Ind. (Mail returned). 1912.  
HECKER, FRANK, 1108 Jefferson Co. Savings Bk. Bldg., Birmingham, Ala.  
HEDLEY, CLARK, Marion, S. C.  
HEDRICK, HORACE A., 2606 Gilford Ave., Baltimore, Md.  
HEER, RUFUS S., 115 4th St., Platteville, Wis.  
HEINY, EDGAR, Hattiesburg, Miss.  
HELMER, JACOB, 327 Madison Ave., Scranton, Pa.  
HEMMY, CHRISTIAN D., New London, Wis. 1910.  
HEMNEBERGER, W. B., Portland, Ore. (Mail returned). 1914.  
HEMPHILL, JOHN F., 428 Dexter St., Clay Center, Kans.  
HENDERSON, LEVI C., Twin Falls, Ida.  
HENDREN, OLIVER T., 6645 Ridge Ave., Philadelphia, Pa.  
HENDREN, S. G., 17 E. Market St., Lewiston, Pa.  
HENNESSY, WILLIAM J., 126 Front St., Worcester, Mass.  
HENRICH, LEO. O., Vacaville, Cal.  
HERBOTT, JULIUS W., 1336 N. Marshall St., Philadelphia, Pa.  
HERNSHEIM, J. T., 6222 Lakewood Ave., Chicago, Ill.  
HERR, T. J., 381 E. 83rd St., New York City.  
HERRING, LAWRENCE JAMES, Wilson, N. C.  
HERRON, MOORE BRYANT, 1282 Pike St., Canonsburg, Pa.  
HERSHEY, CHAS. E., Tiffin, Ohio.  
HERSHEY, SAMUEL E., Lock Box 283, Charleston, W. Va.  
HESS, ORLANDO B., Dept. of Agriculture, Washington, D. C.  
HICKMAN, D. ELWYN, 333 W. Union St., West Chester, Pa.  
HICKMAN, THOMAS S., 1818 Cherry St., Kansas City, Mo.  
HICKS, HAZEN H., 105 Custom House Bldg., San Francisco, Cal.  
HICKS, TUNIS, 642 Irving St., Washington, D. C.  
HICKS, THOS. H., Milbank, S. D. 1911.  
HIDAY, JOHN L., Fortville, Ind.  
HIGGINS, CHARLES H., Experimental Farm, Ottawa, Ont., Can.  
HILL, ANSON HARRIS, Brookings, S. D. 1911.  
HILL, GEORGE H., Atkinson, Ill.  
HILL, JAMES, Tarlac, Tarlac Province, P. I.  
HILL, JAMES ANDERSON, 2609 E. 14th St., Oakland, Cal. 1910.  
HILL, JOSEPH G., Skaneateles, N. Y. 1913.  
HILL, ROBERT C., West Alexandria, Ohio.

- HILL, WM. PROCTOR, Ft. Riley, Kans. (Mail returned). 1912.  
HILLIARD, WILLIAM A., 630 McMillan Ave., Winnipeg, Man.  
HILTON, GEORGE, 126 Lewis St., Ottawa, Ont., Can.  
HILTON, WM., 615 Spence St., Winnipeg, Man.  
HILTY, REUBEN, 619 Walnut St., Toledo, Ohio.  
HINEBAUCH, T. D., Tower City, N. D.  
HINKLEY, C. J., Woonsocket, S. D., Box 43.  
HOAG, WALTER MORROW, 1732 Enterprise St., New Orleans, La.  
1910.  
HOEHN, ALVY M., Ottoville, Ohio. 1912.  
HOEKZEMA, OTTO FREDERICK, McBain, Mich.  
HOGARTY, JOHN J., 1724 Webster St., Oakland, Cal.  
HOGG, EDWIN, 29 Butler Ave., Wilkes Barre, Pa.  
HOLDEN, E. H., Box 612, Springfield, Mass.  
HOLDEN, W. C., 131 No. Camel St., Delphos, Ohio. (Deceased  
April 16, 1916). 1914.  
HOLFORD, FRED DEWITT, Box 655, Chatham, N. Y.  
HOLLANDER, FERDINAND, 4616 Corondelet St., New Orleans, La.  
HOLLINGSWORTH, FREDERICK H., 39 4th St., Council Bluffs, Ia.  
HOLLINGSWORTH, J. B., 105 Cambridge St., Ottawa, Ont., Can.  
HOLLISTER, WM. L., Avon, Ill.  
HOLMES, WALTER BURDETTE, 225 E. Washington St., Springfield,  
Ill.  
HOLT, CAMPBELL L., Box 465, Norfolk, Va.  
HOOD, ARCHIBALD J., 910 Clauranald Ave., Montreal, Que., Can.  
HOOPES, HERBERT, Bel Air, Md.  
HOOVER, LEE C., 11 So. 9th St., Richmond, Ind.  
HOPE, FREDERICK S., 148 N. Paxon St., Philadelphia, Pa.  
HOPPER, JOHN B., 74 Maple Ave., Ridgewood, N. J.  
HOPPER, JOHN G., Chesapeake City, Md. 1914.  
HORNBAKER, JOS. N., Front Royal, Va.  
HORNER, GLENN W., Westminster, Md.  
HORSTMAN, EDWARD, Newton, Miss.  
HOSKINS, CHESTON M., 3452 Ludlow St., Philadelphia, Pa. 1912.  
HOSKINS, H. PRESTON, University Farm, St. Anthony Park, Minn.  
HOUCHIN, A. S., Newark, Del.  
HOUCK, U. G., Washington, D. C.  
HOWARD, C. H., 271 Shelden St., Houghton, Mich.  
HOWARD, CLARENCE T., 11 N. Main St., Sullivan, Ind.  
HOWARD, JULIAN, Stanwood, Wash. (Mail returned). 1910.  
HOWARD, OGDEN JAY, Coloma, Mich. (Mail returned). 1912.  
HOWARD, W. K., Lock Box 43, Gainesville, Tex.  
HOWE, WALTER E., 444 Federal Bldg., Denver, Colo.  
HOYLMAN, JOHN L., Franklin, Nebr.  
HOYMAN, HARRY J., Livestock Exchange Bldg., So. St. Joseph, Mo.  
HUBBELL, ARTHUR D., 318 E. 2nd St., Los Angeles, Cal.  
HUDGINS, PATRICK HENRY, Box 184, Fredericksburg, Va. (Mail  
returned). 1911.

- HUDSON, BENTLEY F., Moweaqua, Ill.  
HUDSON, PERCY WILBUR, Americus, Ga.  
HUEBEN, FRANK W., 1131 Riverview Ave., Kansas City, Kans.  
HUEBSCHMANN, JOHN, 625 S. 3rd St., Baltimore, Md.  
HUELSEN, J., 348 W. 118th St., New York City, N. Y.  
HUFF, LOGAN B., Box 227, Aurora, Ill.  
HUFF, WILSON, 401 West Liberty St., Rome, N. Y.  
HUFFMAN, PLEASANT J., 104-5 Livestock Exchange Bldg., Fort Worth, Texas. 1913.  
HUFNALL, WILLIAM THOMAS, City Food Inspector, Port Arthur, Tex. 1913.  
HUGHES, ARTHUR D., 4193 S. Halstead St., Chicago, Ill. (Deceased Feb. 15, 1916).  
HUGHES, JOSEPH, 2537 State St., Chicago, Ill.  
HUGINS, FRANK ALMON, P. O. Bldg., S. Omaha, Nebr.  
HULL, MARTIN, 20 Federal Bldg., Kansas City, Kans.  
HUMPHREY, EARL H., Santa Maria, Cal. 1914.  
HUMPHREYS, J. C., Chula, Mo.  
HUNT, FRANK, 214 Washington St., Jamestown, N. Y.  
HUNT, J. C., 84 N. Union St., London, Ohio.  
HURD, RAY B., 11th St. and 2nd Ave., South, Payette, Ida.  
HURLEY, PAUL C., East St. Louis, Ill. (Mail returned). 1912.  
HURST, DAN W., Box 218, Tecumseh, Nebr.  
HURST, WILBUR H., Chadron, Neb.  
HURT, LESLIE M., 665 N. Fair Oaks Ave., Pasadena, Cal.  
HUSBAND, AUBREY G., 4th St., Belmont, Man., Can.  
HUTCHISON, JOHN, 7045 Emerald Ave., Chicago, Ill.  
HUTHMAN, G. H., 415 E. 7th St., Portland, Ore.  
HUTTON, JOHN P., East Lansing, Mich.  
HUYETT, WALTER G., East Market St., Wernersville, Pa.  
HYDE, THOMAS F., Brookville, Ind.  
HYLAND, EUGENE H., Schuyler, Nebr.  
HYLTON, FLOYD D., Box 612, Longmont, Colo.  
IDE, ALMOND H., 28 Elm St., Lowville, N. Y.  
IMES, MARION, Federal Bldg., Kansas City, Kans.  
INGRAM, WILLIAM L., Florence, Ala.  
IRWIN, IVAN B., Stonewall, Man. 1912.  
IRWIN, SAMUEL, 24 W. Jackson St., Battle Creek, Mich.  
ISELL, GEORGE P., East 9th St., Hopkinsville, Ky. 1914.  
IVERSON, JOHN P., Box 287, Sacramento, Cal.  
JACKSON, CRAWFORD CHARLES, East Villiard St., Dickinson, N. D.  
JACKSON, FRANK B., Camden, Ohio.  
JACKSON, WILLIAM PETER, 429 Salem St., Chico, Cal.  
JACOB, M., 312 W. Church Ave., Knoxville, Tenn.  
JAFFRAY, DAVID S., JR., 209 N. Des Plaines St., Chicago, Ill.  
JAGO, THOMAS E., Athens, Ga.  
JAKEMAN, HARRY W., University of Nevada, Reno, Nev.  
JAKEMAN, WILLIAM, Glace Bay, Nova Scotia, Can.

- JAMES, THOMAS D., 1123 Washburn St., Scranton, Pa.  
JAMESON, JOHN W., 817 Pleasant St., Paris, Ky.  
JARMAN, G. A., Chestertown, Md.  
JEFFERIES, JOS. R., 1140 Sutter St., San Francisco, Cal.  
JEFFERSON, JOSEPH H., Chicago Jet., Ohio.  
JEFFREY, FRED M., 1890 N. 12th St., Toledo, Ohio.  
JELEN, FRANK, Box 734, Cedar Rapids, Ia.  
JENKINS, ELBERT A., 2027 Morgan St., Shelbyville, Ill.  
JENKS, RALPH C., 78 Croton Ave., Ossining, N. Y.  
JENNINGS, C. G., Morris, Minn.  
JENSEN, H., 2468 E. 28th St., Kansas City, Mo.  
JERVIS, HORACE B., 1205 Lady St., Columbia, S. C.  
JERVIS, JAMES G., 3694 Victoria Drive., Vancouver, B. C.  
JEWELL, CHARLES H., 4th Cavalry, Schofield Barracks, Honolulu, H. T.  
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JOHNSON, ALBERT C., 131 15th St., Portland, Ore. (Mail returned). 1911.  
JOHNSON, AUGUST F., Memphis, Tenn. (Mail returned). 1913.  
JOHNSON, JOSEPH, Union Stock Yards, Lancaster, Pa.  
JOHNSON, LEVI E., 429 Pecks Ave., San Antonio, Tex.  
JOHNSON, OSCAR JOSEPH, State Veterinarian, Miles City, Mont.  
JOHNSTON, NERLS A., 21 Lorne St., Wetaskiwin, Alta.  
JOLLY, CHARLES R., 19 Exchange Place, Atlanta, Ga. 1912.  
JOLY, A., 60 Silver St., Waterville, Maine.  
JONES, ALBERT C., 108 W. Washington St., High Point, N. C.  
JONES, FRANK R., 742 N. Belmont Ave., Indianapolis, Ind. 1912.  
JONES, FREDERIC S., 66th St. and Ave. A., New York City.  
JONES, GEORGE ANDREW, Box 102, Sedro Wooley, Wash.  
JONES, GEORGE B., Sidell, Ill.  
JONES, JAMES M., Lewisburg, Tenn.  
JONES, PHILIP K., 5604 Harvard St., Pittsburg, Pa. 1913.  
JONES, W. F., 311 Second St., E., McCook, Nebr.  
JOPLING, WILLIAM, North Star, Mich.  
JOY, JAMES J., 2436 Collingwood Ave., Detroit, Mich.  
JOSS, JESSE M., 2318 E. 24th St., Kansas City, Mo.  
JOYCE, C. O., Irvington Sta., Ind.  
JUCKNISS, PAUL, 617 N. 23rd St., So. Omaha, Nebr.  
JULIEN, REN C., Delphi, Ind.  
JUNG, OTTO E., 1514 West 30th St., Oklahoma City, Okla.  
JUNGERMAN, GEORGE T., Morrill, Kans.  
KAISER, ALBERT JOHN, Kingsburg, Cal.  
KALKUS, JULIUS, 805 Linden Ave., Pullman, Wash.  
KAMMERER, R. A., 700 South Kingshighway, St. Louis, Mo.  
KANN, R. L., Mechanicsburg, Pa.  
KARTRUDE, EILERT H., Jasper, Minn.  
KATSUNUMA, TOMITZO, U. S. Immigration Service, Honolulu, T. H.  
KAUPP, B. F., West Raleigh, N. C.



- KAY, GUSTAVE A., B. A. I., South Omaha, Nebr.  
KAYLOR, JAMES M., Barry, Ill.  
KEAN, THOMAS J., 1630 W. Suydenham St., Philadelphia, Pa.  
KEANE, CHARLES F., 1818 Market St., San Francisco, Cal.  
KEEF, E. M., Winters, Cal.  
KEEHN, WM. G., 1336 E. 15th St., Kansas City, Mo.  
KEELEY, PETER T., 238 N. Elm St., Waterbury, Conn.  
KEELOR, ALLEN Z., Telford, Pa. 1912.  
KEELOR, J. R., Harleysville, Pa.  
KEENE, HARRY L., Shabbona, Ill.  
KEEPERS, ROBERT W., Greencastle, Pa.  
KELLER, T. O., Ridgeville, Ind.  
KELLY, JAMES SCOTT, 31 Exch. Bldg., Wichita, Kans.  
KELLY, STEPHEN G. C., 1600 S. A. St., Elwood, Ind. 1911.  
KELLY, THOMAS, 1204 Pine St., Philadelphia, Pa.  
KELLY, WM. HENRY, 233 Western Ave., Albany, N. Y.  
KELPE, HENRY O., Box 464, Albuquerque, N. M.  
KELSER, RAYMOND A., B. A. I., Washington, D. C.  
KELSEY, HARRY R., 1344 23rd St., Newport News, Va.  
KENELEY, FRANK THOMAS, Twin Falls, Ida.  
KENNEDY, E. D., Ogden, Utah.  
KENNEDY, JAMES F., Bloomington, Wis.  
KENNEDY, W. W., 74 E. First St., Fulton, N. Y.  
KENNEY, THOS. G., Bismarck, N. D.  
KERSEY, DENNIS L., 15 Bridge St., Danbury, Conn.  
KERN, CHARLES B., Beloit, Kans.  
KERN, HARRY F., B. Agriculture, Manila, P. I.  
KETCHUM, F. D., S. St. Paul, Minn.  
KETTLEHORN, A. H., Broadway, Columbus, Wis.  
KEYS, ARCHIBALD A., 117 N. 10th St., Minneapolis, Minn.  
KICKBUSCH, FRANK O., B. A. I., Grand Rapids, Wis.  
KIERNAN, JOHN A., B. A. I., Birmingham, Ala.  
KIGIN, LAWRENCE C., Lincoln, Nebr. 1914.  
KIGIN, THOMAS F., 102 No. Green St., Tipton, Ind.  
KILLE, WILMER B., 127 Grant St., Salem, N. J.  
KING, JOHN, Carlisle, Sask., Can. 1914.  
KING, SAMUEL A., 1208 Orren St., N. E., Washington, D. C.  
KING, WILLIAM M., 1671 Folsom St., San Francisco, Cal.  
KINGMAN, HARRY E., Fort Collins, Colo.  
KINGMAN, H. W., 19 West Canton St., Boston, Mass.  
KINGSTON, RICHARD H., 41 Convent Ave., New York City.  
KINNEY, WILLIAM M., 133 So. Grant St., Wooster, Wayne Co., Ohio.  
KINSEY, GEORGE W., 931 Market St., Wheeling, W. Va.  
KINSLEY, ALBERT T., 1336 E. 15th St., Kansas City, Mo.  
KINSLEY, CHRISTOPHER C., Oakley, Kans.  
KINYON, B. F., Ladysmith, Wis.  
KIRBY, BASSETT, 85 Cooper St., Woodbury, N. J.  
KIRBY, AMOS COLLINS, Page, N. D.

- KJERNER, RUDOLPH, Chatfield, Minn.  
KLEIN, LOUIS A., 39th and Woodland Ave., Philadelphia, Pa.  
KLINE, A. J., Fulton St., Wauseon, Ohio.  
KLIPPHARDT, WM. A., 3537 A. Wyandotte St., Kansas City, Mo.  
KLOTZ, JOSEPH W., Noblesville, Ind.  
KNAP, ANTON E., Box 1303, Billings, Mont. (Mail returned). 1913.  
KNAPP, ALBERT C., 2414 North Ave., Bridgeport, Conn.  
KNAPP, G. A., Millbrook, N. Y.  
KNAPP, VALENTINE M., 37 New St., Danbury, Conn.  
KNIGHT, RALPH F., 109 W. Henley St., Olean, N. Y.  
KNOWLES, ALBERT D., 302 S. 4th St., W., Missoula, Mont.  
KNOWLES, M. E., Helena, Mont.  
KNOWLES, VIRGIL W., 305 Federal Bldg., Oklahoma, Okla.  
KOCH, JULIUS, East Second St., Downey, Cal.  
KOCHER, FRANK T., 1010 E. Market St., Sunbury, Pa.  
KOEHLER, EDWARD F., 6th and Church Sts., Easton, Pa.  
KOEN, JOHN STRATTON, Adel, Ia.  
KOOON, GEORGE H., 12th Cavalry, Hachita, N. Mex.  
KOONCE, LAFAYETTE F., 324 S. Blount St., Raleigh, N. C.  
KORB, WALTER A., Dept. of Agriculture, Iloilo, P. I.  
KRAGNESS, T. A., 6031 Wentworth Ave., Chicago, Ill.  
KREIDER, W. E., Wadsworth, Ohio.  
KREUZIGER, MARTIN W., Hotel Randolph, Bowman, N. D.  
KREY, THEODORE F., 451 Sheridan Ave., Detroit, Mich.  
KRIEGER, ROBERT E., Williston, N. D. 1911.  
KRON, OSCAR JACOB, 1386 Goldengate Ave., San Francisco, Cal.  
KUBIN, EDISON F., McPherson, Kans.  
KUHN, J. M., Mercersburg, Pa.  
KULP, A. I., Adel, Iowa.  
LACROIX, J. VICTOR, 9 So. Clinton St., Chicago, Ill.  
LADSON, THOMAS ARTHUR, Hyattsville, Md.  
LAMB, CHARLES G., 1140 Corona St., Denver, Colo.  
LAMB, MORGAN B., Dept of Agriculture, Columbus, O. 1911.  
LAMBERT, FREDERICK WILLIAM, New Windsor, Md.  
LAMBRECHTS, T., Box 71, Montevideo, Minn.  
LAMES, G., Dysart, Ia.  
LAMPE, W. H. G., 2939 Broadway, Indianapolis, Ind. 1912.  
LAND, L. M., Limestone and Short Sts., Lexington, Ky.  
LONDON, FRANK D. M., 16 Pleasant St., Great Barrington, Mass.  
LANG, AUGUST R., 152 Main St., Porterville, Cal. (Mail returned).  
1911.  
LANGDON, HARRY B., Charlestown, W. Va.  
LANGFORD, SAMUEL M., College St., Martinsburg, W. Va.  
LANGTRY, WALTER, Fort Wayne, Ind. 1912.  
LAPOINTE, R., St. Peter, Minn.  
LAPPLE, EDWARD, 132 Exch. Bldg., care Northern Serum Co., Sioux  
City, Ia.  
LARGENT, BERT H., Battle Ground, Ind.

- LAROCHE, OMER, 560 Visitation St., Montreal, Can. 1912.  
LARSON, LOUIS N., P. O. Block, Whitehall, Wis.  
LASH, CHRIS. D., care B. A. I., So. St. Joseph, Mo.  
LASSEN, CHRISTIAN W., 516 Bush St., Pendleton, Ore.  
LAUMAN, FREDERICK J., 2300 Cecelia Ave., Sioux City, Ia. 1912.  
LAVIERS, WILLIAM NELSON, Berlin, Ohio. 1912.  
LAW, JAMES, Ithaca, N. Y.  
LAWTON, ANDREW NOBLE, 2 Clinton Ave., Broadhead, Wis. 1914.  
LAWTON, FLETCHER E., 110 Madison St., Greencastle, Ind.  
LAYNE, ERNEST, Huntington, W. Va.  
LEACH, EDWARD DANIEL, 333 Clinton St., Ft. Wayne, Ind.  
LECLAIRE, THOS. EDW., Alberta, Can. P. O. Box 501, Calgary, Alta  
LEE, DANIEL D., 549 Albany St., Boston, Mass.  
LEE, WALTER HERBERT, Brundidge, Ala.  
LEECH, G. EDW., 322 Main St., Winona, Minn.  
LEGENHAUSEN, ADOLPH H., Weldon, Ill. 1913.  
LEGNER, ARTHUR J., Leland, Ill.  
LEIGH, BEVERLY M., 411 20th Ave., Meridian, Miss. 1913.  
LEININGER, DANIEL B., 7th Cavalry, Douglas, Ariz.  
LEITH, FRED J., 1401 Michigan Ave., Chicago, Ill.  
LEITH, THOMAS LESTER, Alliance, Nebr.  
LEMLEY, GEO. GRANT, 2106 Cumming St., Omaha, Nebr.  
LEN, ROBERT C., Mascoutah, Ill. 1912.  
LENFESTY, JOHN H., Lyons, Ohio.  
LENTZ, FRANK E., 39th and Woodland Ave., Philadelphia, Pa. 1911  
LENTZ, WM. J., 39th and Woodland Ave., Philadelphia, Pa.  
LEONARD, MILTON M., 18 South Park Square, Asheville, N. C.  
LEPPLA, L. J., 769 West Monroe St., Chicago, Ill. 1910.  
LESLIE, CHAS. ADELPHUS, 10 Masonic Temple, Deadwood, S. D.  
LETT, HASKELL, 111 W. 3rd St., Seymour, Ind.  
LETTENEY, JAMES T., 42 Charles St., Rochester, N. H.  
LEUTHOLT, HENRY, 250 Main St., Taylor, Pa.  
LEWIS, HAROLD M., 20 Railroad Sq., Nashua, N. H.  
LEWIS, HENRY S., 110 Washington Ave., Chelsea, Mass.  
LEWIS, JAMES, Greenwood, Miss.  
LEWIS, SEYMOUR V., Glenwood City, Wis.  
LEWIS, WALTER KEYS, 901 Union Natl. Bank Bldg., Columbia, S. C.  
LEWIS, WATSON F., Waseca, Minn.  
LICHENWALTER, H. W. C., 1215 15th St., Sacramento, Cal.  
LICHTY, WM. W., Woodstock, Ill.  
LINBERG, JOHN WILLIAM, 228 W. 53rd St., Philadelphia, Pa.  
LINCH, CHARLES, 123 North Allen St., Albany, N. Y.  
LINCOLN WILLIS B., Neuhoft Abattoir & Packing Co., Nashville,  
Tenn.  
LINDBURG, O. ENOCH, 423 N. 10th St., Lincoln, Nebr. 1913.  
LINDLEY, PAUL S., Paoli, Ind. 1912.  
LIPP, CARL FREDERICK, 208 Occidental Life Bldg., Albuquerque,  
N. M.

- LIPP, CHARLES C., Brookings, S. D.  
LIPP, GEORGE A., Box 818, Roswell, N. M.  
LOBDELL, STEPHEN C., 6 S. 3rd St., LaFayette, Ind.  
LOCKE, GEORGE H., Lockford, Cal.  
LOCKETT, STEPHEN, Univ. of Nev., Reno, Nev.  
LOCKHART, ANDREW A., Carnduff, Sask.  
LOGAN, EDWARD A., 3209 Mitchell Ave., St. Joseph, Mo.  
LOGAN, JAMES AUSTIN, Oakes, N. D.  
LOLLAR, ERNEST E., Red Cloud, Nebr.  
LOMBARD, CHARLES M., 4502 Emerald Ave., Chicago, Ill.  
LONGLEY, OTIS A., 24th and Broadway, Oakland, Cal. Western  
Laboratories.  
LOTHE, HERBERT, Waukesha, Wisconsin.  
LOUCK, REX CLARK, Clarence, Ia.  
LOVE, GROVER V., Main St., Chalmers, Ind.  
LOVE, JAMES R. (address unknown). 1910.  
LOVEBERRY, CLARENCE, Quartermaster Dept. U. S. Army, San Francisco, Cal. 1910.  
LOVEJOY, J. O., Federal Bldg., Augusta, Ga.  
LOVELAND, GROVE W., 49 East Main St., Torrington, Conn.  
LOVELL, ROY, 200 W. 5th St., York, Nebr.  
LOWE, J. PAYNE, 171 Jefferson St., Passaic, N. J.  
LOWE, W. S., 17 North 1st St., Phoenix, Ariz. 1912.  
LOWREY, FREDERICK H. S., 1127 Keele St., Toronto, Ont., Can.  
LUEDER, CHARLES AUGUSTUS, 752 North Front St., Morgantown,  
W. Va.  
LUKES, HARRY, 441 Sumner Ave., Springfield, Mass.  
LULL, ELMER L., Parma, Ida.  
LUTHER, W. H., Boonville, Ind.  
LUZADOR, ROY A., Morrisonville, Ill.  
LYMAN, RICHARD P., P. O. Box 1018, East Lansing, Mich.  
LYON, H. C., Hutchinson, Minn.  
LYTLE, W. HARRISON, Salem, Ore.  
MCALLISTER, RANCIE G., 1259 Fillmore St., Corvallis, Ore.  
MCADORY, ISAAC S., Auburn, Ala.  
MCALPINE, D., Box 696, Brockville, Ont., Can.  
MCANULTY, JOHN F., 2832 North 6th St., Philadelphia, Pa. 1912.  
MCCAFFREY, JAMES, Lock Box 83, Red Bank, N. J.  
MCCAIN, EARL A., Gregory, S. D. 1912.  
MCCARTHY, CHAS. F., 1317 Fulton St., San Francisco, Cal. 1912.  
MCCARTHY, F. H., 317 N. 3rd St., Pottsville, Pa.  
MCCARTHY, HENRY J., Arlington, Md.  
MCCARTHY, THOS. A., Livestock Exch. Bldg., Chicago, Ill. (Mail  
returned). 1914.  
MCCARTNEY, JOHN, Middletown, N. Y.  
MCCASKILL, ALEXANDER WHITNEY, Weyburn, Sask., Can.  
MCCAUGHEY, N. W., Church St., Veterinary Hospital, Presque Isle,  
Me.



- McCLAIN, L. GORDON, Manzanola, Colo. 1910.  
McCLELLAN, MILTON PORTER, 222 Hongus Crescent, Regina Sask.  
McCLELLAND, ALFRED H., Walton, N. Y.  
McCLELLAND, FRANK E., 455 Ellicott St., Buffalo, N. Y.  
McCLOSKEY, ANTHONY J., Chestnut Hill, Pa.  
McCORD, FRANK A., 215 Queens Ave., Edmonton, Alta.  
McCOWEN, D., Edgeley, N. D.  
McCOY, ELLIS E., Canton, Miss.  
McCOY, FRANKLIN C., 1623 South I St., Bedford, Ind.  
McCOY, JOHN E., Box 21, Cawker City, Kans.  
McCRANK, J. A., 79 Saily Ave., Plattsburg, N. Y.  
McCUAIG, D., McAdam Junction, New Brunswick, Can.  
McCULLEY, ROBERT W., 38 Lexington Ave., New York City, N. Y.  
McCULLOUGH, EDW. ALEX., 219 McDowell St., Delavan, Wis.  
McCURDY, FRANK C., 616 No. 10th St., St. Joseph, Mo.  
McCUSHING, FRANCIS P., 104 High St., Keene, N. H.  
McDANIEL, J. C., 1319 S. A. St., Elwood, Ind.  
McDANIEL, JOHN SAMUEL, East Lansing, Mich.  
McDONALD, D. M., 3932 Pleasant Ave., Minneapolis, Minn.  
McDONNELL, L. E., Audubon, Minn.  
McDONOUGH, JAMES, 47 Portland Place, Montclair, N. J.  
McDONOUGH, JOHN F., 1633 So. 22nd St., Philadelphia, Pa.  
McDOWELL, CLARENCE, 614 2nd St., Watertown, S. D. 1911.  
McDOWELL, HARRIS B., Middletown, Del. 1912.  
McELYEA, LEWIS WESLEY, Ames, Ia.  
McEVERS, ALBERT E., 349 Michigan Ave., Chicago, Ill. (Mail returned). 1911.  
McFARLAND, C. M., 230 Exch. Bldg., Sioux City, Ia.  
McFATRIDGE, HOWARD SIMONA, 30 Argyle St., Halifax, N. S., Can.  
McGILVRAY, CHAS. D., Dept. of Agriculture, Winnipeg, Man., Can.  
McGILLIVRAY, GEORGE, Box 303, Roseau, Minn.  
McGROARTY, BERNARD, Boothwyn, Pa. 1913.  
McGUIRE, W. C., Pitt and Second Sts., Cornwall, Ont., Can.  
McINNES, BENJAMIN KATER, 57 Queen St., Charleston, S. C.  
McKAY, ALEXANDER M., 527 4th Ave., Calgary, Alta.  
McKENNA, JOHN F., 616 Eye St., Fresno, Cal.  
McKENZIE, K. J., Northfield, Minn.  
McKEON, WM. JOS., 716 Cambie St., Vancouver, B. C. 1910.  
McKERCHER, ARTHUR, 115 Ionia St., E., Lansing, Mich.  
McKEY, JOHN, 1926 W. 1st St., Duluth, Minn.  
McKIBBIN, DAVID JR., 2900 Frankfort Ave., Philadelphia, Pa.  
McKILLIP, CHESTER A., 1639 Wabash Ave., Chicago, Ill.  
McKILLIP, GEORGE B., 1639 Wabash Ave., Chicago, Ill.  
McKILLIP, MATTHEW H., 1639 Wabash Ave., Chicago, Ill.  
McKILLIP, WALTER J., 1639 Wabash Ave., Chicago, Ill.  
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McKIM, ORVILLE ERNEST, 338 West 56th St., New York City.  
McKINNEY, WM. J., 585 Driggs St., Brooklyn, N. Y.

- McKINNON, JOHN ALEX., Land Transportation, Manila, P. I.  
McLAIN, JOHN H., Inkster, N. D.  
McLEAN, ADAM T., Truro, Nova Scotia, Can.  
McLEAY, JOHN DONALDSON, Indianapolis, Ind.  
McLEOD, J. H., Charles City, Ia.  
McMULLEN, ROBERT H., 444 Federal Bldg., Denver, Colo.  
McNAIR, FREDERICK H., 2126 Haste St., Berkeley, Cal.  
McNALLY, MICHAEL, 1412 Pine St., St. Louis, Mo. 1914.  
McNAUGHTON, D. D., 717 Kelly Ave., Devil's Lake, N. D.  
McNEIL, JAMES C., 3349 Webster Ave., Pittsburg, Pa. 1910.  
McNEIL, JOHN H., Sao Paulo, Brazil, S. America. (Mail returned).  
1914.  
McPIKE, CLARENCE T., Cando, N. D.  
MACCORMACK, C. DOUGLAS, North Baltimore, Ohio.  
MACDONALD, R. W., 522 Brush St., Flint, Mich. (Mail returned).  
1911.  
MACINTOSH, ROBERT DUNCAN, 296 Danforth Ave., Toronto, Can.  
(Mail returned). 1914.  
MACK, C. A., Gilbert Plains, Man., Can.  
MACK, JAMES F., River Falls, Wis.  
MACK, WINFRED B., Univ. of Nevada, Reno, Nev.  
MACKELLAR, ROBERT S., 351 West 11th St., New York, N. Y.  
MACKELLAR, WM. M., 219 Federal Bldg., San Diego, Cal.  
MACKIE, CLEMENT L., Towson, Md. (Mail returned). 1913.  
MACKIE, FRANK H., 1035 Cathedral St., Baltimore, Md.  
MADSON, WM., Appleton, Wis.  
MAHAFFY, THOMAS J., Box 920, Jacksonville, Fla.  
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MALCOLM, PETER, New Hampton, Ia.  
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MALONE, WILLIAM J., Mt. Horeb, Wis.  
MANGAN, DANIEL J., 2557 Third Ave., Bronx, New York City.  
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MANUEL, EDWARD A., 232 Park Pl., Des Plaines, Ill.  
MARKS, DANIEL G., 562 West 12th St., Chicago, Ill. 1912.  
MARQUARDT, S. J., Barnesville, Ohio.  
MARQUETTE, WILLIAM M., 240 Hudson St., Indianapolis, Ind. 1912.  
MARQUIS, FRANCIS, M., French Camp, Cal.  
MARSH, HADLEIGH, 3401 16th St., N. W., Washington, D. C.  
MARSHALL, CHARLES WILLIAM, Main St., Brewster, N. Y.  
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MARSHALL, HENRY, 202 Federal Bldg., Richmond, Va.  
MARSHALL, HERBERT, Mechum River, Va.  
MARSHALL, L. G., Towanda, Pa. 1911.  
MARSTELLER, ROSS P., College Station, Texas.

- MARTIN, HARRY D., 481 Rhode Island St., Buffalo, N. Y.  
MARTIN, ROBERT D., 1192 Broad St., Bridgeport, Conn.  
MARTIN, STANLEY THOMAS, 281 James St., Winnipeg, Man., Can.  
MARTIN, W. E., 281 James St., Winnipeg, Man., Can.  
MARVEL, ALEX. L., Owensville, Ind.  
MASON, A. L., Oahu, T. H.  
MASON, WILLIAM DENT, Box 488, Elko, Nev.  
MATHEWS, E., 187 Grand St., Jersey City, N. J.  
MATROCCE, DANIEL, J. St., Los Banos, Cal.  
MATTSON, WM. H., Chester Heights, Pa. 1912.  
MAULDIN, COLUMBUS E., Jeanerette, La.  
MAULFAIR, CHAUNCEY D., R. D. No. 2, Granville, Ill. 1910.  
MAWER, GEORGE C., 9219 Clifton Blvd., Cleveland, Ohio.  
MAXSON, WILBUR BERNETT, Flemington, N. J.  
MAYNARD, LEE H. P., 1937 Market St., Philadelphia, Pa.  
MAYO, N. S., 4753 Ravenswood Ave., Chicago, Ill.  
MAZE, LUTHER A., care Parke, Davis & Co., Rochester, Mich.  
MEAD, R. N., Crawfordsville, Ind.  
MEADE, ALBERT M., National Stock Yards, Ill. (Mail returned).  
1912.  
MEADORS, W. H., National Stock Yards, E. St. Louis, Ill.  
MEADS, FREDERICK F., 123 E. Randolph St., Enid, Okla.  
MEAGHER, JOHN A., Box 172, Glendale, Ohio. 1912.  
MEBANE, WM. LONG, Rockwood, Maine  
MEGOWAN, CLAUDE L., 1021 J. St., Sacramento, Cal.  
MEINERS, R. F., Boonton, N. J. 1914.  
MEISNER, H. A., 1133 Hartford Ave., Baltimore, Md.  
MEIXEL, GEORGE A., Aurora, Nebr. 1911.  
MERILLAT, LOUIS A., 1827 Wabash Ave., Chicago, Ill.  
METCALFE, A. R., Van Kleek Hill, Ont., Can.  
MEYER, GEORGE W., 528 Washington St., New York City.  
MEYER, KARL F., Second and Parnassus Ave., San Francisco, Cal.  
MEYERHOEFFER, JOS. STEWART, Weyers Cave, Va. 1911.  
MEYST, FREDERICK W., 144 Bryant St., N. W., Washington, D. C.  
MICHAEL, LEO B., East Main St., Collinsville, Ill.  
MICHAEL, WM. ROBERT, Highland, Ill.  
MICHNER, HIRAM M., North Wales, Pa. 1914.  
MIDDLETON, ASA C., Grundy Center, Ia.  
MILKS, HOWARD J., N. Y. State Veterinary College, Ithaca, N. Y.  
MILLARD, HUGH R., 2507 Central Ave., Cheyenne, Wyo.  
MILLEN, CHARLES J., care Jacob E. Decker & Sons, Mason City, Ia.  
MILLER, A. DALE, 29 Vine St., Dayton, Ohio. 1913.  
MILLER, A. W., 4534 S. 18th St., Omaha, Nebr.  
MILLER, C. A., 411 South Jackson St., Louisville, Ky.  
MILLER, CHESTER, Elliott & Co., Duluth, Minn.  
MILLER, DANIEL S., 230 Mill St., East Port Chester, Conn.  
MILLER, FREDERICK A., Fitchburg, Mass.  
MILLER, HARRY K., 128 W. 53rd St., New York City.

- MILLER, JOHN FRED, 24 New Scotland Ave., Albany, N. Y.  
MILLER, JOHN M., 152 Louis St., Grand Rapids, Mich.  
MILLER, JOHN P., 46 Reed St., Reading, Pa. 1913.  
MILLER, SAMUEL H., 2125 3rd Ave., Rock Island, Ill. 1910.  
MILLER, THOMAS C., 600 Tribune Bldg., Winnipeg, Man., Can.  
MILLS, C. C., 355 E. Main St., Decatur, Ill.  
MILLS, H. LEE, 118 Pearl St., Burlington, Vt. 1913.  
MINER, GEORGE H., 180 Claremont Ave., Buffalo, N. Y.  
MISSALL, FRANK CHARLES, Cor. Mill and Pine Sts., Santa Maria, Cal.  
MITCHELL, ADRIAN J., SR., 1219 Peach St., Erie, Pa. 1914.  
MITCHELL, AQUILA, 3rd Field Artillery, Fort Sam Houston, Tex.  
MITCHELL, GEORGE C., Klamath Falls, Ore.  
MITCHELL, HARRY BARTLE, R. F. D. 2., Wellsboro, Pa.  
MITCHELL, JAMES FRANCIS, Anaconda, Mont. Hotel Montana.  
MITCHELL, J. R., 610 S. 3rd St., Evansville, Ind. 1912.  
MITTERLING, IRA, 302 Montgomery St., Hollidaysburg, Pa.  
MIX, C. C., 110 W. Jackson St., Battle Creek, Mich.  
MOCK, WM., 53 N. Fourth St., Easton, Pa.  
MOEGLING, RICHARD EMIL, 3017 Jefferson Ave., Cincinnati, Ohio.  
1913.  
MOHLER, JOHN R., Dept of Agriculture, Washington, D. C.  
MOLGARD, PETER C., Ruskin Nebr.  
MOLT, FRED S., Big Spring, Tex.  
MOOBERRY, OLIVE WAKON, Morton, Ill. 1912.  
MOODY, ARTHUR H., 402 Armitage St., Three Rivers, Mich.  
MOODY, ROBERT P., 111 Sutton St., Maysville, Ky.  
MOORE, A. E., 175 Waverly St., Ottawa, Can.  
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MOORE, HAMLET, 610 N. Rampart St., New Orleans, La.  
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MOORE, SHEARD, 608 Iberville St., Donaldsonville, La.  
MOORE, VERANUS A., New York State Vet. College, Ithaca, N. Y.  
MOORHOUSE, WM. B., 37 Main St., Tarrytown, N. Y. 1913.  
MOREHOUSE, WALTER G., 746 Mill St., Salem, Ore.  
MOREL, JULES F., State Board of Health, Portland, Ore. (Mail returned). 1912.  
MOREY, B. FRANKLIN, 235 S. Main St., Clinton, Ind.  
MORGAN, FRANK W., 6th and Cherry Sts., Chattanooga, Tenn.  
MORGAN, WM. JEROME, Seaton, Ill.  
MORIN, C. L., 9 Messenger St., St. Albans, Vt.  
MORRIS, EDWARD H., 221 Main St., Derby, Conn.  
MORRIS, HARRY, Experiment Station, Baton Rouge, La. 1913.  
MORROW, ALBERT CAINE, Dillon, Mont.  
MORSE, JOHN H., Box 377, Sumter, S. C.  
MOSS, HARRY T., 710 W. Third St., Dayton, Ohio.  
MOUNT, WILLET C., 1155 Walnut St., Red Bluff, Cal.  
MOYER, B. FRANKLIN, 3929 Baltimore Ave., Philadelphia, Pa.  
(Mail returned). 1912.



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MOYER, FRANK LEONARD, Carey, Ohio.  
MOYER, VINCENT C., South Hampton, Pa. (Mail returned). 1914.  
MUELLER, FERDINAND A., 459 E. Washington St., Indianapolis, Ind.  
MULLER, J. HERMAN, Long Green, Md.  
MULVEY, CHARLES J., Mooers, New York.  
MUNCE, THOMAS W., R. D. No. 2, Washington, D. C. (Mail returned).  
MUNCE, T. EDWARD, State Livestock Sanitary Board, Harrisburg, Pa.  
MUNGER, GRANT B., 2226 N. Illinois St., Indianapolis, Ind.  
MUNN, ALBERT A., Kearney, Nebr.  
MUNN, AHIZAH, J., Fayette, Mo.  
MURCH, ALFRED LITTLEFIELD, 17 Boynton St., Bangor, Me. 1913.  
MURISON, JAMES J., Manor St., Arcola, Sask., Can.  
MURPHEY, HOWARD SYLVESTER, 519 Welch Ave., Ames, Iowa.  
MURPHY, BERNARD W., 604 N. 11th St., St., Joseph, Mo.  
MURPHY, DANIEL E., Prairie due Sac, Wis.  
MURPHY, FRANCIS P., 1813 N. 2nd St., Philadelphia, Pa. 1914.  
MURPHY, JOSEPH WHITMORE, Columbia, S. C.  
MUSSELMAN, S. F., Frankfort, Ky.  
MYDLAND, GUSTAV HERMAN, Everest Kans.  
MYERS, HARRY E., 140 S. Main St., Fostoria, Ohio.  
MYERS, M. J., Ft. Payne, Ala.  
MYERS, SIDNEY D., 215 Walnut St., Wilmington, Ohio.  
MYERS, W. F., Fort Wayne, Ind. (Deceased).  
NANCE, JOSEPH E., Anadarko, Okla.  
NASH, ELMER DENNETT, 717 Sixth Ave., Helena, Mont.  
NATTRESS, JOSEPH T., Delevan, Ill.  
NAYLOR, RALPH EDELEN, Cheyenne, Wyo., Box 397.  
NEILSON, NORMAN, 345 Market St., Colusa, Cal.  
NEBEKER, SHIRLEY, Lake Town, Utah. 1912.  
NEFF, S. C., Staunton, Va. 1912.  
NELSON, AMOS F., 656 E. 21st St., Indianapolis, Ind.  
NELSON, CHARLES A., 224 Front St., Brainard, Minn.  
NELSON, CONRAD L., Box 691, S. St. Joseph, Mo. 1913.  
NELSON, NELSON L., Ames, Ia.  
NELSON, S. B., Pullman, Wash.  
NEUHAUS, CHARLES O., Union Stock Yards, Pittsburgh, Pa.  
NEWBURG, LOUIS, 2442 Forest Ave., Kansas City, Mo.  
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NEWCOMER, E. W., Mt. Joy, Pa.  
NEWGENT OTTAWAY C., 1448 Sycamore St., Terre Haute, Ind.  
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- NEY, WILLIAM O., Michigan City, Miss.  
NICE, FRANK K., 3514 N. 15th St., Philadelphia, Pa.  
NICHOLAS, GEORGE B., 1404 Holmes St., Kansas City, Mo. 1914.  
NICHOLS, PERCIVAL K., 107 Harrison Ave., Port Richmond, N. Y.  
NIGHBERT, E. M., 211 Federal Bldg., Atlanta, Ga.  
NIGHBERT, JAMES D., Pittsfield, Ill.  
NILES, W. B., Box 927, Ames, Ia. 1912.  
NIMPHIUS, HARRY F., 761 E. 163rd St., New York City.  
NISSLEY, SOLOMON M., 18 Spring St., Bellefonte, Pa.  
NIVEN, ANDREW B., Inspector in charge Hog Serum Station, Henderson, Ky.  
NIXON, ROBERT B., Demopolis, Ala.  
NOACK, OTTO G., 54 S. 6th St., Reading, Pa.  
NOBACK, CHARLES V., Otisville, N. Y.  
NOBLE, GEO. EDW., 1312 Grove St., Boise, Idaho.  
NOONAN, ALBERT J., Bernard, Ia. 1913.  
NORDEN, CARL J., 1518 Pasco St., Kansas City, Mo.  
NÖRGAARD, VICTOR A., Honolulu, Hawaii, Territorial Veterinarian.  
NORRIS, CLARENCE L., 4 L. S. Record Bldg., U. S. Stockyards, Chicago, Ill.  
NORTHROP, LEONARD E., 105 N. Davidson St., Indianapolis, Ind.  
NORTON, J. C., Cor. Washington and 1st Ave., Phoenix, Ariz.  
NORTON, OSCAR M., 109 Main St., Greenville, Miss.  
NORTON, ROBERT S., Velva, N. D.  
NOYES, ORRIN W., Valentine, Nebr.  
NULPH, PEARLEY E., Crosby, N. D.  
NUNN, HENRY, Box 337, McMinnville, Ore.  
O'BANION, ARCHIE L., Box 439, Santa Barbara, Cal.  
O'BRIEN, PAT. JOS., 732 S. Figueroa St., Los Angeles, Cal. 1912.  
O'BRYAN, SHERID, Pesotum, Ill.  
O'CONNOR, JOSEPH, West Hope, N. D.  
O'DONNELL, MICHAEL J., 241 Grove St., Blue Island, Ill.  
O'HARRA, WILLIAM G., Galloway, Ohio.  
ODELL, EDWIN O., Central City, Nebr.  
OESTERHAUS, JOHN H., 465 Livestock Exchange, Kansas City, Mo.  
OLIVER, WALTER GORDON, 840 2nd St., San Diego, Cal.  
OLSEN, LUTHER E., care U. S. B. A. I., So. St. Paul, Minn.  
OLTHOUSE, MARTIN, Grass Lake, Mich.  
O'NEAL, WM., Newman, Cal.  
O'REILLY, JAMES M., 508 E. 3rd St., Merrill, Wis.  
O'ROURKE, MICHAEL JOHN, 720 Valencia St., San Francisco, Cal.  
ORME, FRANK W., 720 Valencia St., San Francisco, Cal. 1910.  
ORME, THOS. WHITFIELD, 260 5th St., San Bernardino, Cal. 1910.  
ORR, WILLIAM CROSBY, Dillon, Mont.  
ORTIZ, CARLOS, 26 Villa St., Ponce, Porto Rico. 1913.  
OSBORN, ORIN H., Paynesville, Minn.  
O'TOOLE, STEPHEN, Agricultural College, Fargo, N. D.  
OUTHIER, C. B., Salinas, Cal.

- PACE, JOHN C., Box 597, Calexico, Cal.  
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PAINE, HAROLD, Rosetown, Saskatchewan,  
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PALMER, DONALD B., Live Stock Sanitary Board, St. Paul, Minn.  
PALMER, FLOYD ELBERT, Owatonna, Minn.  
PALMER, H. F., R. D. No. 2, Parma, Mich.  
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PAQUIN, LEON A., Box 225, Webster, Mass.  
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PARKER, LEON L., 439 Main St., Catskill, N. Y.  
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PARSE, FRANKLIN L., Columbia, Miss.  
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PATRICK, MURRAY E., Penn Ave., Greenburg, Pa.  
PATTERSON, ELIJAH E., 650 Grand River Ave., Detroit, Mich.  
PATTERSON, E. I., Mound City, Mo.  
PATTERSON, HENRY G., 915 Gravier St., New Orleans, La. 1913.  
PATTISON, HOMER D., Box 40, Beloit, Wis.  
PAUL, ARTHUR, Box 291, Portersville, Cal.  
PAULSEN, THOMAS C., 506 Government St., Baton Rouge, La. 1910.  
PAXSON, WM. H., Marietta, Pa.  
PAXTON, IRVING B., Box 562, Red Bluff, Cal.  
PEARCE, CHARLES D., 10 Carhart Ave., Binghamton, N. Y.  
PEARCE, DEE, Box 222, Celeste, Tex.  
PEARCE, FRANK H., Carson, Ia. (Mail returned).  
PEARSON, CHARLES, Amarillo, Tex.  
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PECK, SANFORD ARTISAN, Oak Grove, Mo.  
PEDERSON, GUNERIUS M., Hamler, Ohio.  
PEIRCE, HARRIE W., 83 Playstead Road., West Medford, Mass.  
PEIRCE, LAWRENCE L., 10 Central St., Arlington, Mass.  
PENNIMAN, G. P., Cor. Exchange and Commercial Sts., Worcester, Mass.  
PERKINS, CHESTER R., 19 Hillside Ave., Buffalo, N. Y.  
PERRIGO, W. H., 580 1st Ave., Milwaukee, Wis. 1912.  
PERRY, CHAS. H., 82 Park Ave., Worcester, Mass.  
PERRY, F. M., Edgell St., Framingham, Mass.  
PERRY, JAMES G., Box 294, Ennis, Tex.  
PETERS, A. T., Box 43, Peoria, Ill.  
PETERSEN, THEODORE J., Ukiah, Cal. 1914.  
PETERSON, W. E., 16 Lyman St., Waltham, Mass.  
PETHICK, W. H., Charlottetown, P. E. Isle, Can.  
PETTY, CLARENCE C., Lake Odessa, Mich.  
PFARR, ALBERT WM., 4623 Friendship St., Pittsburg, Pa.

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PHELPS, OLIVER JAY, Mill St., Lexington, Ky., Y. M. C. A. Bldg.  
PHILIPS, CHAS. S., Mt. Vernon, Wash.  
PHILLIPS, J. M., 3732 West Pine Blvd., St. Louis, Mo.  
PHILLIPS, S. C., Sheridan, Ind. (Mail returned). 1914.  
PHILPOTT, LUTHER B., 248 West 4th St., Provo, Utah. 1911.  
PHILP, FRED W., Box 204, Mineral Point, Wis.  
PHYFE, WALTER H., 9 Grove St., Middletown, N. Y.  
PIATT, D. A., 1706 2nd Ave., Birmingham, Ala.  
PICKENS, EARL MAX, 106 Cook St., Ithaca, N. Y.  
PIELEMEIER, HENRY, R. D. No. 1, Solsberry, Ind.  
PIERCE, FOSTER H., Faulkton, S. D. 1914.  
PIERRET, WILBUR, B. A. I., So. St. Joseph, Mo.  
PIKE, FREDERICK, 817 1st Ave., Spokane, Wash. 1910.  
PINE, HENRY E., Carrizozo, N. M. 1914.  
PINKROSS, ROLF D., Camp Dennison, Ohio. 1913.  
PIRIE, LESLIE D., 172 Garfield St., Santa Cruz, Cal.  
PISTOR, ADOLPH H., B. A. I., Washington, D. C.  
PLANTZ, JOHN FRANK, 50 E. Buchtel Ave., Akron, Ohio.  
PLATT, ROBERT M., Coldwater, Kans.  
PLAYDON, C. H., Reading, Mass.  
PLUMMER, ALEX., 1140 Sutter St., San Francisco, Cal.  
POE, C. E., 113 E. Franklyn St., Hagerstown, Md.  
POLLARD, JOHN SAMUEL, 183 Harrison St., Providence, R. I.  
POMFRET, HENRY, 158 Athole St., Winnipeg, Man., Can. 1914.  
POOLEY, JOHN R., 820 S. 14th St., E., Cedar Rapids, Ia.  
POPE, GEORGE W., 1340 Meridian Place, N. W., Washington, D. C.  
POPE, LEMUEL, JR., Orleans, Mass. 1912.  
PORTER, E. C., 120 N. Mercer St., New Castle, Pa.  
PORTER, FRED W., 104 N. Ross Ave., Tampa, Fla.  
POTE, THOMAS B., 4925 Park View Place., St. Louis, Mo.  
POTTER, GEO. M., B. A. I., Washington, D. C.  
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POWERS, MAURICE RAY, Norwalk, Conn.  
PRESLER, H. A., Fairbury, Ill.  
PRESTON, MARVIN JAMES, Moosomin, Sask. (Mail returned). 1914.  
PRICE, CHAS. E., 310 East 5th St., Santa Ana, Cal. 1911.  
PRICE, JOHN O. F., Morris & Co., Memphis, Tenn.  
PRIEN, OTTO LOUIS, Box 626, Laramie, Wyo. (Mail returned). 1912.  
PRIEN, ROLAND H., R. F. D. No. 31, Morgan Hill, Cal. 1912.  
PRIEST, BENJ. H., 501 Flower St., Kern Branch, Cal.  
PRIOR, ROBERT, 214 S. 2nd St., North Yakima, Wash. 1912.  
PRITCHARD, JOHN W., Fessenden, N. D.



- PRITCHARD, WM. T., North Platte, Nebr.  
PRITCHETT, LAMAR F., Auburn, Ala.  
PROUSE, HARVEY LEE, Allen, Nebr.  
PROUSE, WM. C., 615 4th Ave., Minneapolis, Minn.  
PRUCHA, JOSEPH V., care Dr. Howe, Denver, Colo.  
PUGH, WALTER MAURICE, Powers Lake, N. D.  
PUGH, WM. T., 57 Elm St., South Bridge, Mass.  
PULLAM, JOHN H., Santa Ana, Cal.  
PULVER, WM. A., Wamego, Kans.  
PURCELL, JNO. T., Federal Bldg., Rapid City, S. D.  
PURDY, MARION ALEXANDER, 621 10th St., Shelbyville, Ky.  
QUIGLEY, ROSCOE MURPHY, Cor. Logan Ave. and Herold St., Ty-  
rone, Pa. 1914.  
QUIN, ABNER H., Creston, Ia.  
QUINN, JAMES E., Antioch, Cal.  
QUINN, P. E., 632 W. Washington St., Indianapolis, Ind.  
QUINN, THOS. F., 709½ 9th St., Greeley, Colo. 1910.  
QUITMAN, E. L., 1514 West VanBuren St., Chicago, Ill.  
RADCLIFF, BURDETT D., 108 Franklin Square, New Britain, Conn..  
RADER, EDWARD B., Clinton, Wis. 1910.  
RAFTER, EDWARD, Hamburg, N. Y.  
RAGAN, JAMES A., 510 Liberty St., Morris, Ill.  
RAGLAND, MARCUS J., Salisbury, N. C.  
RAMSEY, R. A., B. A. I., Washington, D. C.  
RAMSEY, SAMUEL A., JR., 234 Third St., Terre Haute, Ind. 1913.  
RAMSEY, SAMUEL V., 234 South Third St., Terre Haute, Ind.  
RAMSAY, WM., J. C., Box 481, Watsonville, Cal.  
RANCK, EDWARD M., Agricultural College, Miss.  
RANSOM, SHERMAN, 1923 3rd St., Vancouver, B. C. (Mail re-  
turned). 1913.  
RAQUE, CHAS. A., 219 Federal Bldg., Spokane, Wash.  
READ, H. W., 27 Court St., Freehold, N. J.  
READHEAD, WM., Lenox, Ia.  
REGAN, W. J., 606 River St., Patterson, N. J.  
REARDON, JOHN D., Bureau of Agriculture, Manila, P. I. 1913.  
REBER, ABRAM N., 2104 N. Tremont St., Kansas City, Kans.  
REBOLD, GEORGE P., 4553 Wabash Ave., Chicago, Ill. (Mail re-  
turned). 1911.  
RECORDS, EDWARD J., Univ. of Nevada, Reno, Nev.  
REDHEAD, WM. H., Dept. of Health, Cleveland, Ohio. 1914.  
REED, RAYMOND C., Newark, Del.  
REEFER, LEON N., 1405 Chapline St., Wheeling, W. Va.  
REICHEL, JOHN, care H. K. Mulford Co., Glenolden, Pa.  
REICHMANN, ANDREW FRANCIS, Armour, S. D.  
REICHMANN, FERDINAND A., Geddes, S. D.  
REID, WILLIAM, Yorkton, Sask.  
REIFSNYDER, IRVIN S., Collegeville, Pa.  
REIHART, OLIVER F., 835 N. 22nd St., S. Omaha, Nebr.

- RENO, JOHN S., Southport, Ind.  
RENTER, ELMER J., 767 Delhi Ave., Cincinnati, Ohio.  
RENTER, WALTER W., 2632 W. Sixth St., Cincinnati, Ohio.  
RENTSCHLER, MANDON D., 232 N. Front St., Punxsutawney, Pa.  
REVERCOMB, GEO. ARCHIE, Lewisburg, W. Va.  
REY, CHARLES R., Tulare, Cal.  
REY, GEORGE S., S. Court St., Visalia, Cal.  
REYNOLDS, FRANCOIS H. K., B. A. I. Quarantine Div., Washington, D. C.  
REYNOLDS, HOWARD C., Factoryville, Pa.  
REYNOLDS, M. H., Experimental Farm, St. Paul, Minn.  
RHEA, R. LEE, Houston & Bowie Sts., San Antonio, Tex. 1913.  
RHODAS, WARREN L., Lansdowne, Pa. 1913.  
RHODES, C. J., Beloit, Wis.  
RICE, JOHN M., Cambridge St., S., Lindsay, Ont., Can.  
RICE, RAY D., Maple Rapids, Mich.  
RICEBARGER, BENJAMIN F., 200 W. Main St., St. Charles, Ill.  
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RICH, THEODORE S., 1477 West Grand Blvd., Detroit, Mich.  
RICHARDS, THOS. H., 1127 Keele St., Toronto, Ont., Can.  
RICHARDS, WM. R., care W. N. Neil, So. Omaha, Nebr. 1914.  
RICHARDS, WILLIAM WILSON, Manila, P. I.  
RIDDELL, ROBERT, 460 Chester St., Victoria, B. C.  
RIDGE, WILLIAM H., Maple Ave., Somerton, Philadelphia, Pa.  
RIEDEL, PHILIP H., 1401 Roache St., Indianapolis, Ind.  
RIETZ, J. H., 633 Crawford Ave., Ames, Ia.  
RIKE, HARRY W., 735 Linden Ave., Burlingame, Cal. (Deceased).  
RILEY, EDWARD H., Experiment Station, Bozeman, Mont.  
RIORDON, J. J., Beverly Farms, Mass. 1914.  
RIORDON, WM. F., Gilroy, Cal.  
RISHEL, ALBERT E., care U. S. Consulate, Liverpool, Eng.  
RISLEY, HARRY B., 74 Adams St., Brooklyn, N. Y.  
RITTER, PHILIP, 1609 East 37th St., Kansas City, Mo.  
RITTER, ROY W., Holtville, Cal.  
RIVERS, REUBEN N., Lansdale, Pa. 1913.  
ROACH, FRANK, Box 489, Pendleton, Oregon.  
ROADHOUSE, CHESTER L., College of Agriculture, Berkeley, Cal.  
ROBERTS, GUY A., Agricultural Experiment Station, W. Raleigh, N. C.  
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ROBERTSON, JAMES, 735 East 44th St., Chicago, Ill.  
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ROBINSON, JOHN W., Garrison, N. D.

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ROBINSON, PAUL L., 535 Public Ave., Beloit, Wis. 1913.  
ROBINSON, THOMAS E., 65 Main St., Westerly, R. I.  
ROBINSON, WM. B., 72 Maysville St., Mt. Sterling, Ky.  
ROBINSON, W. H., 87 Leland St., Portland, Maine.  
ROCKWELL, ARCHIE MERRITT, Eleanor, Ill.  
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ROME, JOHN, Germantown, Md.  
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ROSENBERGER, ARTHUR C., Box 1091, Stockton, Cal.  
ROSENBERGER, GUY W., San Diego, Cal.  
ROSENBERGER, MAYNARD, 219 Federal Bldg., San Diego, Cal.  
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ROSS, JAMES D., 796 William Ave., Winnipeg, Man., Can.  
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ROUB, J. F., Monroe, Wis.  
ROUECHE, R. C., 1382 W. 73rd St., Cleveland, Ohio.  
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RUNDLE, THOMAS THEODORE, 435 First St., Oxnard, Cal.  
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RUNYON, PETER F., 24 E. Main St., Freehold, N. J.  
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RUTHERFORD, J. G., Canadian Pacific Railway Co., Calgary, Alta.  
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RYAN, FRANK C., Middletown, Md.  
RYAN, J. F., 2525 Indiana Ave., Chicago, Ill. 1913.  
RYDER, HERMAN R., 5719 W. Superior St., Chicago, Ill.  
RYDER, J. E., Forest Hills Inn, Forest Hills Garden, L. I., N. Y.  
SADLER, ERNEST D., Wagner, S. D.  
SALLADE, J. W., Auburn, Pa.  
SALSBERY, C. E., 4220 Chestnut St., Kansas City, Mo.  
SANDERS, ALFRED E., 1033 Shelby St., Indianapolis, Ind. 1914.  
SANDERSON, WILLIAM, Sidney, Ohio.  
SANFORD, E. F., 115 Sterling Place, Brooklyn, N. Y.

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SAVAGE, ARTHUR J., 414 E. Pikes Peak Ave., Colo. Springs, Colo.  
SAVAGE, WILLARD A., Box 527, Tucumcari, N. M.  
SAWYER, F. N., Bakersfield, Cal.  
SAYRE, B. HARRY, Brookings, S. D.  
SCHADER, CURTIS H., Sunnyside, Wash.  
SCHAEFER, EDW. H., 3215 Anderson Ave., Kansas City, Mo. 1914.  
SCHAEFER, G. L., Tekamah, Nebr.  
SCHAEFER, VALENTIN, Tekamah, Nebr.  
SCHAEFFTER, E. P., 408 P. O. Bldg., Detroit, Mich.  
SCHALK, ARTHUR F., Agricultural College, N. D.  
SCHAUFLE, CHAS. A., 1338 Hunting Park, Philadelphia, Pa. 1912.  
SCHERMERHORN, ROBERT J., 106 E. Citrus Ave., Redlands, Cal.  
SCHLEICH, FRED HARRISON, Williamsport, Ohio.  
SCHLOEMER, CHAS. C., 602 River Terrace, Hoboken, N. J.  
SCHMIDT, HUBERT, College Station, Tex.  
SCHNEIDER, ERNEST, Kulm, N. D.  
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SCHNEIDER, FRANCIS O., Nicholasville, Ky. Box 504.  
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SCHROEDER, E. C., B. A. I., Experiment Station, Bethesda, Md.  
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SCHWARZE, HERMAN R., 500 S. Douglas Ave., Springfield, Ill.  
SCHWARTZ, JOHN A., Lawrence, Ind. 1912.  
SCHWARZKOPF, OLAF, Fort Sam Houston, Tex.  
SCHWEIN, PAYSON E., Elkhart, Ind.  
SCOTT, CARL J., Knoxville, Ia.  
SCOTT, GEO. A., Waterloo, Iowa.  
SCOTT, JOHN, 236 Eaton St., Peoria, Ill.  
SCOTT, JOHN A., Waverly, Minn. 1913.  
SCOTT, M. W., 24 S. 6th St., Vincennes, Ind.  
SCOTT, WM. A., 1407 1st Ave., Columbus, Ga.  
SEELEY, JAMES T., No. 1101 Westlake Ave. N., Seattle, Wash.  
SEIBERT, WALTER D., 808 Howard St., Petoskey, Mich. 1913.  
SELBY, ORVAL C., Worthington, Minn.  
SELF, PRESLEY M., Farmersburg, Ind.  
SENSEMAN, BENJ. F., 1723 N. 55th St., Philadelphia, Pa.  
SEVERCOOL, LUCIUS A., 217 Lake Ave., Elyria, Ohio.  
SEVERIN, JOHN R., 2014 So. Olive St., Sioux City, Iowa.  
SEVEROVIC, MIRKO F., 1833 Blue Island Ave., Chicago, Ill.  
SEVENSTER, JOHN, Washington St., Hamburg, Ia.  
SEXMITH, A. B., Charlotte, Mich.



- SEXTON, MICHAEL J., 819 Marquette Ave., Minneapolis, Minn.  
SHAFFER, DALLAS W., 4035 Olive St., St. Louis, Mo. 1910.  
SHAND, JAMES REID, U. S. Army, Tientsin, China.  
SHARP, WALTER E., Newton, Ia.  
SHARTLE, WALTER W., 413 N. New Jersey St., Indianapolis, Ind.  
SHAW, CHARLES W., 200 W. 78th St., New York City.  
SHAW, CLARENCE E., 115 Sterling Place., Brooklyn, N. Y.  
SHAW, WILLIAM HARVEY, Pawnee, Ill.  
SHEALEY, ALONZO S., Bureau of Agriculture, Manila, P. I.  
SHEARBURN, THOS. O., Walnut, Ill.  
SHELDON, THOS., Rhinebeck, N. Y. 1911  
SHELTON, J. E., Box 308, Arkansas City, Kans.  
SHEPARD, E. H., 2027 E. 105th St., Cleveland, Ohio.  
SHEPPARD, WM., Neck Road, Sheepshead Bay, Long Island, N. Y.  
SHERIDAN, GEORGE, Ashland, Nebr.  
SHERWOOD, ARTHUR M., Naperville, Ill.  
SHEVALIER, EUGENE D., Escanaba, Mich.  
SHIGLEY, RALPH E., Kenmare, N. D.  
SHIKLES, ERNEST ADAIR, Dearborn, Mo.  
SHILLINGER, JACOB E., Easton, Md.  
SHIPLEY, LEVI U., Sheldon, Ia.  
SHIPLEY, TRAJAN, Live Stock Exchange, Sioux City, Ia.  
SHIPMAN, OLIVER B., Hilo, T. H.  
SHONYO, JOHN HOWARD, 352½ Portage Ave., Winnipeg, Man., Can.  
SHORE, CHALMER S., Lake City, Minn.  
SHORE, HOWARD J., Dept. of Agriculture, Washington, D. C.  
SHREVE, RAYMOND M., Vine and Church Sts., Panora, Ia.  
SHULER, C. C., Eaton, Ind.  
SHUMWAY, DANIEL G., B-4 Carlton Court, Buffalo, N. Y.  
SHUTTE, VERNON D., Volcano House, Kau, T. H.  
SIGLER, THOS. A., 114 N. Jackson St., Greencastle, Ind.  
SIGMOND, CHAS. J., Box 80, Pipestone, Minn.  
SILFVER, OSCAR, Norwood, La.  
SILVERWOOD, HERBERT, 331 Clackmas St., Portland, Ore.  
SIHLER, C. J., 7th and Everett Ave., Kansas City, Kans.  
SIMMS, BENNETT THOMAS, Oregon Agricultural College, Corvallis,  
Ore.  
SIMMONS, WILLIAM HERBERT, 2344 W. Chestnut St., Louisville, Ky.  
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SIMPSON, HAL C., Box 69, Denison, Iowa.  
SIMPSON, WM. H., 45 Dartmouth St., Malden, Mass.  
SIMS, FRANK C., Aurora, Nebr.  
SIMS, THOS., Woodburn, Ore.  
SISSON, SEPTIMUS, Ohio State University, Columbus, Ohio.  
SKERRITT, HENRY W., 315 Columbia St., Utica, N. Y.  
SKINNER, JOHN FREDERICK, Box 167, Morris, Man., Can.  
SLATER, J. HARVEY, 3012 Holmes St., Kansas City, Mo.  
SLATER, LEROY E., 1115 Prospect St., Indianapolis, Ind.

- SLAWSON, ALEXANDER, 248½ St. Nicholas Ave., New York City. 1912.
- SLOULIN, G. E., Aneta, N. D. 1912.
- SMEAD, MORGAN J., Parkedale Farm, Rochester, Mich.
- SMELLIE, JAMES, Eureka, Ill.
- SMITH, A. W., Farmer City, Ill.
- SMITH, BERT C., Brigden, Ont., Can.
- SMITH, CLARENCE E., Dept. of Public Health, Greenville, S. C.
- SMITH, FLETCHER EUGENE, 4801 Evanston Ave., Seattle, Wash.
- SMITH, GEORGE F., Vicksburg, Miss. 1914.
- SMITH, GEO. W., 157 6th St., Hoboken, N. J.
- SMITH, HERBERT M., 407 Federal Bldg., Providence, R. I.
- SMITH, HENRY V., 90 Wildmere Ave., Detroit, Mich.
- SMITH, JESSE P. F., 2310 Central Ave., Kansas City, Kans.
- SMITH, ROBERT P., Wendell, Ida.
- SMITH, R. V., 17 Court St., Frederick, Md.
- SMITH, STANLEY N., 112 College Ave., Columbia, Mo.
- SMITH, S. P., Cando, N. D.
- SMITH, T. E., 309 Barrow St., Jersey City, N. J.
- SMITH, WM. B., Interlaken, N. Y. 1911.
- SMITH, WRIGHT J., 260 Clinton Ave., Kingston, N. Y.
- SMYTHE, FRANK R., 1523 Groesbeck Rd., Cincinnati, Ohio.
- SNYDER, RUDOLPH, 444 Federal Bldg., Denver, Colo.
- SOCKMAN, CLIFFORD CLYDE, Deshler, Ohio.
- SOLLBERGER, R. J., 1412 S. 8th St., St. Louis, Mo.
- SONGER, LEE C., Dept. of Agriculture, Olathe, Kans.
- SOLT, C. H., Arlington, Ohio.
- SORENSEN, ANDREAS I., R. F. D. No. 6, Stockton, Cal.
- SORRELL, WARREN, Willard, N. M.
- SPADE, FRED A., Constantine, Mich.
- SPALLING, N. C., 760 W. Center St., Somerset, Colo.
- SPEAR, WM. H., 122 Cumberland Ave., Portland, Me.
- SPENCER, H. F., 1415 J. St., Sacramento, Cal. 1910.
- SPENCER, H. H., 429 W. Adams St., Jacksonville, Fla.
- SPENCER, TRACY N., 7 W. Depot St., Concord, N. C.
- SPRAGUE, JOHN D., Seward, Nebr.
- SPRINGER, C. W., 48 Morgantown St., Uniontown, Pa.
- SPRINGER, SAMUEL E., B. A. I., S. Omaha, Neb.
- SPRINGER, U. S., 27-29 Hastings St., bet. Canal and Ottawa, Grand Rapids, Mich.
- STAAB, JOHN J., 1422 W. 65th St., Cleveland, Ohio.
- STALEY, RAYMOND M., Camp Hill, Pa.
- STAMP, ASHLEY G., 198 Candler Ave., Highland Park, Mich.
- STANCLIFT, RAY J., 15th Cavalry, Fort Riley, Kans.
- STANFORD, JOHN F., Fayetteville, Ark. 1911.
- STANGE, C. H., Iowa State College, Ames, Ia.
- STAPLES, WM. D., 1813 Quintard Ave., Anniston, Ala.
- STATES, HARRY E., 93 Bagley Ave., Detroit, Mich. 1912.

- STEDDON, R. P., 1468 Harvard St., Washington, D. C.  
STEEL, EDWARD R., Chester, Nebr.  
STEELE, HARRY F., Ft. McIntosh, Laredo, Tex.  
STEELE, JOSEPH G., 217 Exchange Bldg., Ft. Worth, Tex.  
STEHLE, FREDERICK, JR., 5145 Pine St., Philadelphia, Pa.  
STEINBACH, FREDERICK G., Oak and Pacific Ave., Wildwood, N. J.  
STEPHENS, RUSSELL A., B. A. I., Cincinnati, Ohio. 1910.  
STEPHENS, GEORGE, 18 Maplewood Terrace, White River Jct., Vt.  
STETSON, HENRY G., 24 Cliff St., St. Johnsbury, Vt.  
STEVENS, CECIL H., Box 106, Stevensville, Mont.  
STEVENS, GUY G., 20 Park St., Groton, N. Y.  
STEVENS, HERBERT L., 192 Limerock St., Rockland, Me.  
STEVENSON, GEORGE T., Burlington, Vt. 1913.  
STEVENSON, JAMES A., Gretna, Man.  
STEWART, C. E., Chariton, Ia.  
STEWART, H. L., Chariton, Ia.  
STEWART, SAMUEL L., 3335 Brooklyn Ave., Kansas City, Mo.  
STEWART, WALTER C., West Union, Ia.  
STEWART, WALTER J., 4352 Montgomery St., Oakland, Cal.  
STICKEL, WILLIAM E., Etna Mills, Cal.  
STINER, JARVIN O., Lindsay, Cal.  
STINSON, WILLIAM, Chelsea, Mass. 1910.  
STIVER, M. B., Elgin, Man. 1911.  
STOKES, WILFRED J., Ft., Riley, Kans.  
STONE, GARRY T., Sidney, N. Y.  
STOUDER, KIRK W., Ames, Ia.  
STOUFFER, DAVID F., Bellevue, Nebr.  
STOVER, JOHN PRICE, Shady Grove, Pa.  
STRAYER, JOSEPH EDW., Hartington, Neb.  
STREETS, JNO. JAMES, Box 87, Ventura, Cal.  
STRIBLING, WM. E., New England, N. D.  
STRICKLER, C. M., 25 Elm Lane, Greencastle, Pa.  
STRINGER, N. I., Lock Box 195, Stewartville, Minn. 1912.  
STRODTMAN, OTIS E., 211 E. 5th Ave., Arkansas City, Kans. 1912.  
STROUP, WM. L., Corinth, Miss.  
STRUTHERS, CHAS. E., Union Stock Yards, Chicago, Ill. (Mail returned). 1912.  
STUBBS, EVAN LEE, State Farm, R. D. 3., Media, Pa.  
STUBBS, GEORGE W., Opelousas, La. 1910.  
SUGG, REDDING S., 243 Market St., Washington, N. C.  
SULLIVAN, JAMES, 807 N. Eldorado St., Stockton, Cal. 1911.  
SULLIVAN, WALTER A., Box 666, Twin Falls, Ida.  
SUMMERFIELD, JAS. J., Main and First Sts., Santa Rosa, Cal.  
SUNDERVILLE, EARL, Forest Home, Ithaca, N. Y.  
SUTTER, ERROLD, Beach, N. D. 1913.  
SUTTON, OTIS L., 1701 Kinney Ave., Cincinnati, Ohio.  
SWENERTON, L. DANIEL, 500 8th Ave., E., Vancouver, B. C. 1913.  
SWENSON, SIGWART R., Maddock, N. D.

- SWITZER, WILLIAM B., 50 East 7th St., Oswego, N. Y.  
SYLVESTER, JOHN FLETCHER, Langdon, N. D.  
TADE, JAMES M., 518 N. 1st St., Vincennes, Ind.  
TALBERT, JOSEPH F., 721 W. 8th St., Kansas City, Kans.  
TALBOT, PERCY R., Box 703, Edmonton, Alta.  
TAMBLYN, DAVID S., Box 616, Regina Sask., Can. 1914  
TANSEY, EDWARD J., Monrovia, Ind.  
TAYLOR, CHAS. H., 632 Sycamore Rd., DeKalb, Ill.  
TAYLOR, GEO. C., Redding, Cal.  
TAYLOR, LAWRENCE L., Condon, Ore.  
TAYLOR, WALTER J., Dos Palos, Cal.  
TEIE, JOHN A., Hatton, N. D.  
TENCHNICK, DERK, 1639 Wabash Ave., Chicago, Ill. 1912.  
TENNENT, J. H., 275 King St., London, Ont., Can.  
THACKER, THOMAS, Renfrew, Ont., Can. 1913.  
THOMPSON, CHAS. GOFF, 204 E. Main St., Little Falls, N. Y. 1910.  
THOMPSON, JOHN A., Bureau of Agriculture, Manila, P. I.  
THOMPSON, JOHN B., Harvey, N. D.  
THOMPSON, JOHN S., 903 East 5th St., Moscow, Ida.  
THOMPSON, JOSHUA P., 16 Grand Ave., N. Billings Mont. 1911.  
THOMPSON, MULFORD C., Box 145, Sharon, Conn.  
THOMPSON, WARWICK M., 1210 Main St., Red Bluff, Cal.  
THOMPSON, WILLIAM, Box 145, Laredo, Tex.  
THOMAS, RAYMOND F., Canton, Miss. 1914.  
THORNTON, EDWARD L., Fort Kent, Me.  
TIEFENTHALER, FRANK, Cambridge City, Ind. 1912.  
TILLMAN, ALBERT C., Earlville, Ill.  
TIMMONS, WILFRED H., Box 254, Madison, Ind.  
TIPTON, WILLIAM B., Emporia, Kans.  
TODD, ROBERT S., New Milford, Conn.  
TOLMIE, S. F., Box 1518, Victoria, B. C.  
TOMLINSON, W. J., Williamsport, Pa. 1914.  
TOMPKINS, LELAND J., Walton, N. Y.  
TOOLEY, JAS. WM., 38 4th St., Fond du Lac., Wis.  
TORGERSON, HENRY E., Gilroy, Cal.  
TOPMILLER, ALEXANDER C., P. O. Box 224, Murfreesboro, Tenn.  
TORRANCE, F., Veterinary Director-General, Ottawa, Ont., Can.  
TOTTEN, G. E., 410 Fulton Bldg., Pittsburg, Pa.  
TOW, EDWARD, 1415 J. St., Sacramento, Cal. 1914.  
TOWNE, GEORGE V., Box 54, Thompson, Conn.  
TOWNER, ALBERT N., Towners, N. Y.  
TOWNSEND, GEORGE, New Glasgow, N. S., Can.  
TOWNSEND, NORRIS L., 104 W. 42nd St., New York City.  
TRACY, ANGUS W., Sherbrook, Que., Can.  
TRAINOR, PETER F., 42 Mercer St., Jersey City, N. J. 1913.  
TRAUM, JACOB, Univ. of California, Berkeley, Cal.  
TREADWAY, CHARLES R., 2438 Prospect Ave., Kansas City, Mo.  
TRICKETT, ARTHUR, 1336 E. 15th St., Kansas City, Mo.

- TRIGG, WILLIAM STARK, Aiken, S. C.  
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TRUAX, BLAIR W., Burr Oak, Kans.  
TURLINGTON, JOHN A., Melfa, Va.  
TURNER, H. A., Rose Ave., Pleasanton, Cal.  
TURNER, HENRY W., New Hope, Pa.  
TURNER, JOHN E., 204 S. Detroit St., Kenton, Ohio.  
TURNER, J. P., 916 O. St., N. W., Washington, D. C.  
TUTTLE, CHAS. D., Canton, S. D. 1910.  
TUTTLE, LYFORD E., Grafton, N. H.  
TUXILL, A. J., 5 Lincoln St., Auburn, N. Y.  
TYLER, JOHN LOU, 125 S. Main St., Pomona, Cal.  
TYNER, ALPHEUS L., R. R. 4, Kempton, Ind.  
UDALL, D. H., N. Y. State Vet. College, Ithaca, N. Y.  
UNDERHILL, B. M., 3 West 3rd St., Media, Pa.  
UTT, JAMES GARFIELD, 721 G. St., Sacramento, Cal.  
VAIL, IRVING ROE, 20 George St., New Haven, Conn.  
VAIL, WALLACE F., 267 Greenwich Ave., Greenwich, Conn. 1910.  
VANS DE ERE, JACOB, Sherwood, N. D.  
VANS AGNEW, ROBERT, Ft. Leavenworth, Kans.  
VAN EENENAM, JOHN, Salem, S. D. 1910.  
VAN ES, L., M. D., Agricultural College, Fargo, N. D.  
VEIT, WM., 240 N. 52nd St., Philadelphia, Pa.  
VELDHUIS, ZACHARY, 408 P. O. Bldg., Detroit, Mich.  
VENZKE, HARRY E., Garretson, S. D. (Mail returned). 1912.  
VERMILYA, RALPH F., Minnehaha Apartment No. 25, St. Paul, Minn.  
1913.  
VIGNEAU, JOSEPH H., Union Market Hotel, Watertown, Mass.  
VLEIT, GEO. B., 202 Baldwin St., Hackettstown, N. J.  
VOLLMER, CARL G., Bryans, Ohio.  
VOORHEES, E. R., 87 E. Main St., Somerville, N. J.  
VULLIAMY, H. F., Plaqueimine, La. 1914.  
WADDLE, GEORGE, Kalamazoo, Mich. 1912.  
WAGAMAN, GROVER M., 210 W. Superior St., Kokomo, Ind.  
WAGNER, CHARLES W., Elkton, Ky.  
WAGONER, C. OTTO, 121 S. 10th St., Richmond, Ind.  
WALCH, CLEMENCE C., B. A. I., Exchange Bldg., S. St. Joseph, Mo.  
WALCH, CHAS. IRA, 4823 King Hill Ave., St. Joseph, Mo.  
WALKER, R. G., 238 Aberdeen St., Chicago, Ill. 1914.  
WALKLEY, SEYMOUR J., 185 Northwestern Ave., Milwaukee, Wis.  
WALMSLEY, F. D., 924 Ruttyer St., Utica, N. Y.  
WALTERS, PERCY KNIGHT, Suite 11, Argyle Court, Calgary, Alta.  
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WALSH, L. S. N., 4225 Delmar Blvd., St. Louis, Mo.  
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WARD, GEORGE R., 424 Persia Ave., San Francisco, Cal.  
WARD, HARRY C., Fulton, Mo.



- WARD, JOHN E., 12 E. Crescent St., Grand Rapids, Mich.  
WARD, S. H., State Capitol, St. Paul, Minn.  
WARD, VICTOR, Paso Robles, California.  
WARNER, CHAS. G., 701 S. 4th St., Padukah, Ky. 1913.  
WARNOCK, DAVID, House of Commons, Ottawa, Ont., Can.  
WASHBURN, HENRY J., 704 B. St., S. W., Washington, D. C.  
WASHBURN, W. B., 172 E. Market St., Tiffin, Ohio.  
WATERS, R. E., 28 Village Rd., Gravesend, Long Island, N. Y.  
1913.  
WATSON, EDWARD A., Box 567, Lethbridge, Alta.  
WATSON, THOS. W., Kerens, Tex.  
WAUGH, JAMES A., 1100 Fifth Ave., Pittsburgh, Pa.  
WAY, CASSIUS, 108 Hudson St., New York City.  
WEBB, A. J., 2347 Hudson Ave., Ogden, Utah.  
WEBB, JAMES CLEVELAND, Piedmont, Ala.  
WEBB, WILLIAM T., Quarryville, Pa.  
WEBB, W. W., Auburn, Ala.  
WEBBER, CARR R., 135 Crossman Terrace, Rochester, N. Y.  
WEBBER, ORRIN B., 69 Front St., Rochester, N. Y.  
WEBER, HENRY STANLEY, 6128 Park Ave., Weehawken, N. J.  
WEBER, JOHN H., Boise, Ida.  
WEBSTER, JOHN H., Y. M. C. A., Reno, Nev.  
WEBSTER, WILLIAM OWENS, Utica, Ohio.  
WEEKS, CORNELIUS C., 309 3rd St., N. W., Washington, D. C.  
WEHLE, FRANK A., 586 Sayre Ave., Lexington, Ky.  
WEIGEL, MARION S., Cromwell, Ind.  
WEGNER, EARL EDWARD, Pullman, Wash.  
WEINMAN, JOSEPH E., Arcadia, Neb.  
WEIR, ROBERT, 84 Grove St., Rutland, Vt.  
WEITZEL, FRED, 100 Parkway West., Pittsburg, Pa.  
WELCH, GUY NOBLE, 39 Union St., Northfield, Vt.  
WELCH, JOHN, Rolland, Man. 1911.  
WELCH, WM. B., 353 W. Arrow St., Marshall, Mo.  
WELLS, THOMAS GROVER, Arthur, Ill. 1914.  
WENDE, BERNARD P., 101 Florence Ave., Buffalo, N. Y.  
WENDE, HORATIO S., Tonawanda, N. Y. 1914.  
WERNTZ, HARVEY GRANT, E. Liberty, P. O. Box 321, Pittsburg, Pa.  
WERSHOW, MAX, Veterinary Clinic Bldg., Columbus, Ohio.  
WERTZ, SIDNEY S., Kenesaw, Nebr.  
WESTCOTT, GEORGE F., 1008 Congress St., Portland, Me.  
WESTCOTT, HENRY B., 1008 Congress St., Portland, Me.  
WEST, JAY P., 121 Monona Ave., Madison, Wis.  
WESTERHEIDE, EDWARD FRANCIS, Minster, Ohio. 1912.  
WESTGATE, SAMUEL S., Box 1763, Grafton, N. D.  
WHEELER, A. S., Biltmore, N. C.  
WHITCOMB, MORTON S., Livestock Sanitary Board, St. Paul, Minn.  
WHITE, D. S., 1656 Neil Ave., Columbus, Ohio.  
WHITE, ERNEST A., 1233 Dryade St., New Orleans, La.

- WHITE, GEORGE R., 1314 Adams St., Nashville, Tenn.  
WHITE, JOHN L., 5327 Union Ave., Chicago, Ill.  
WHITE, LOGAN A., Sioux Rapids, Ia.  
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WHITE, V. C., Blackfoot, Ida.  
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WHITESTINE, ORVILLE G., 47 E. Washington St., Huntington, Ind.  
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WHITNEY, HARRISON, 20 George St., New Haven, Conn.  
WHITNEY, J. C., 3 West St., North, Hillsdale, Mich.  
WHITNEY, JOHN GREGORY, 26 Summer St., Montpelier, Vt.  
WHITTLESEY, R. TSALL, 714 E. 7th St., Los Angeles, Cal. 1911.  
WHYTE, JOHN D., 315 Sixth Ave., E., Calgary, Alta.  
WIGHT, W. E., 237 McKee Place, Pittsburg, Pa.  
WICKS, A. G., 23 N. College St., Schenectady, N. Y.  
WILEY, MORRIS C., Trinidad, Colo., B. A. I.  
WILKINS, JOHN E., Johnson and Jordan Sts., Greenville, Tex.  
WILL, EVAN J., Harrisonburg, Va.  
WILLETT, FREDERICK C., Henry, Ill.  
WILLIAMS, GEO. M., 611 College St., Boone, Ia.  
WILLIMAN, EARL LEROY, Ohio City, Va.  
WILLIS, HARRY S., Orange, Va.  
WILLS, J. G., 27 Matilda St., Albany, N. Y.  
WILLYOUNG, LESTER E., 11th Cavalry, Ft. Oglethorpe, Ga.  
WILSON, CLAUD, 113 So. State St., Greenfield, Ind.  
WILSON, FRED O., 314 Madison St., Greenbay, Wis.  
WILSON, JOHN OSCAR, Box 714, Miles City, Mont.  
WILSON, ROBERT H., care Parke, Davis & Co., Rochester, Mich.  
WILTRANT, FRANK ADAM, 16 Washington St., Wilkes Barre, Pa.  
WING, CHARLES C., McCloud, Cal. 1914.  
WINSLOE, J. A. H., Cooperstown, N. D.  
WINSLOW, C., Rockland, Mass.  
WINSLOW, JOSEPH H., Lamoure, N. D.  
WINSTANLEY, JOHN H., 119 S. 37th St., Philadelphia, Pa.  
WINTER, HOWARD E., 132 E. Second St., Plainfield, N. J.  
WINTERINGHAM, HARRY BARKER, 118 Western Ave., Petaluma, Cal.  
WIPF, J. D. C., Belgrade, Mont.  
WISE, WILLIAM F., 246 W. Liberty St., Medina, Ohio.  
WISNER, SCOTT, Omega, Neb.  
WITMER, HERVEY W., Bradentown, Fla.  
WITTE, CHARLES R., New Britain, Conn. 1913.  
WOLCOTT, LEROY B., Shelton, Nebr.  
WOLCOTT, WALTER A., 713 East Johnson St., Madison, Wis.  
WOLF, ORTHO C., West 7th St., Ottawa, Kans.  
WOOD, A. L., Hampton, Ia.

- WOOD, EZRA P., 415 Market St., Charlottesville, Va.  
WOOD, FREDERICK WM., Cutter Laboratory, Berkeley, Cal.  
WOOD, PAUL EWING, Ottawa, Ohio. 1912.  
WOODEN, MORRIS, Wardman Court, Washington, D. C. 1912.  
WOODLIFFE, MARK J., 637 E. 20th St., Denver, Colo.  
WOODSIDE, JAMES H., Auburn, Wash.  
WOODWARD, B. T., Pennsylvania Ave and 28th St., S. E., Washington, D. C.  
WOOLFOLK, GEO. H., Pottsville, Pa.  
WORCESTER, HARRY, 118 W. 3rd St., Middletown, Ohio.  
WORMS, ALBERT C., 2932 Broadway, Chicago, Ill.  
WRAY, A. M., Denver, Colo.  
WRIGHT, C. C., Health Bureau, Portland, Ore.  
WRIGHT, LESLIE A., Water St., Columbus, Wis.  
WRIGHT, NORMAN, 137 20th St., West Saskatoon, Sask.  
WRIGHT, W. DEAN, 1227 Missouri Ave., Portland, Oregon.  
WURM, JOHN E., Pigeon, Mich.  
YANCEY, WILLIAM E., 864 S. Virginia St., Reno, Nev.  
YARD, WILLIAM W., Hotel Ayers, Denver, Colo. 1913.  
YOUNG, GEORGE D., 9945 S. Irving Ave., Chicago, Ill.  
YOUNG, G. R., 4602 Center St., Omaha, Neb.  
YOUNG, HULBERT, 515 N. Charles St., Baltimore, Md.  
YOUNG, JOHN M., 419 6th St., Brooklyn, N. Y.  
YOUNG, WILLIAM A., Prospect, N. Y.  
YOUNGBERG, STANTON, Bureau of Agriculture, Manila, P. I.  
YUNKER, ELKAN H., 2344 N. 18th St., Philadelphia, Pa.  
ZEILER, JOHN LEWIS, Orosi, Tulare Co., Cal.  
ZELL, CHARLES AUGUST, 4362 Kenmore Ave., Edgewater Station, Chicago, Ill.  
ZICKENDRATH, ERNEST G., 101 Middlefield, Road, Palo Alto, Cal.

**THE NUMBER OF VETERINARIANS IN ACTIVE PRACTICE IN EACH  
STATE AND THE MEMBERSHIP OF THE AMERICAN  
VETERINARY MEDICAL ASSOCIATION COMPARED  
WITH THE VALUE OF THE LIVESTOCK**

(Compiled by the Secretary of the American Veterinary Medical  
Association, June, 1916).

STATE	No. of Practicing Veterinarians*	No. of A.V.M.A. Members	Percentage of A.V.M.A. Members	Total Value Livestock†	Value of Livestock per Veterinarian
Alabama	150	22	14.6	\$65,595,000	\$ 437,300
Ariz. and N. Mexico	49	18	36.7	69,546,000	1,419,306
Arkansas	213	3	1.4	74,058,000	347,690
California	371	140	37.7	127,600,000	343,935
Colorado	80	32	40.0	70,161,000	877,012
Connecticut	109	33	30.2	14,164,000	129,944
Delaware	16	4	25.0	6,817,000	426,062
Dist. of Columbia	25	44	176.0	153,000	6,120
Florida	47	5	10.6	20,591,000	438,106
Georgia	69	10	14.4	80,394,000	1,165,130
Idaho	71	15	21.1	49,775,000	701,056
Illinois	919	126	13.7	308,805,000	336,022
Indiana	725	107	14.7	173,860,000	239,806
Iowa	650	78	12.0	393,003,000	604,620
Kansas	570	53	9.4	253,524,000	444,778
Kentucky	264	21	7.9	117,487,000	445,026
Louisiana	59	22	37.2	44,699,000	757,610
Maryland	118	30	25.4	32,570,000	276,016
Massachusetts	450	55	12.2	20,741,000	46,091
Michigan	445	68	15.2	137,804,000	309,671
Minnesota	306	55	17.9	161,641,000	528,238
Mississippi	132	21	15.1	75,247,000	570,053
Missouri	351	71	20.2	285,839,000	814,356
Montana	68	21	30.8	85,663,000	1,259,750
Maine	84	13	15.4	25,162,000	299,547
Nebraska	349	69	19.7	222,222,000	636,739
Nevada	15	10	66.6	19,214,000	1,280,933
New Jersey	165	36	21.8	24,589,000	149,024
New York	637	152	23.7	183,091,000	287,427
North Carolina	92	15	16.3	62,650,000	680,978
North Dakota	158	61	38.6	108,250,000	685,126
New Hampshire	58	9	15.5	11,910,000	205,344
Ohio	682	105	15.3	197,332,000	289,343
Oklahoma	288	8	2.7	152,433,000	529,281
Oregon	83	22	26.5	59,462,000	716,409
Pennsylvania	548	154	26.2	141,480,000	258,175
Rhode Island	33	6	18.1	3,276,000	99,272
So. Carolina	281	15	5.3	45,131,000	160,608
So. Dakota	180	31	17.2	127,229,000	706,827
Tennessee	173	14	8.1	110,706,000	639,919
Texas	437	33	7.5	318,647,000	729,169
Utah	31	9	29.0	28,782,000	928,451
Virginia	103	27	26.2	74,891,000	727,097
Vermont	83	12	14.4	22,643,000	272,807
Washington	125	29	23.2	48,865,000	390,920
West Virginia	251	13	5.1	43,336,000	172,653
Wisconsin	461	59	12.7	158,529,000	343,880
Wyoming	38	6	15.7	65,606,000	1,726,473

\*Includes only those veterinarians in each state who have registered this year under the Harrison Narcotic Act, presumably all in active practice. Probably this does not include many municipal, state or federal employees or veterinarians connected with laboratories.

†From Year Book, U. S. Dept. of Agriculture, 1914, P. 642.

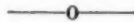
## SOCIETY MEETINGS

### BRITISH COLUMBIA VETERINARY ASSOCIATION

A public meeting on matters relating to the public health was held under the auspices of the British Columbia Veterinary Association in the Board of Trade Rooms, Victoria B. C. on Friday, May 19th, 1916, and was well attended both by the general public and the aldermen, councillors, and health officials of the city and surrounding districts.

The programme consisted of address of welcome by the President, Dr. S. F. Tolmie, explaining the objects of these public meetings. The first address was by Dr. Knight on Dairy Inspection in its relation to the Public Health. The second address was by Dr. Jagger entitled the Relation of the Veterinarian to the Public and to the Stockman. This was followed by an address by Dr. Jervis, illustrated by lantern slides made by himself, on meat inspection and its value to the public health, pointing out the limitations of the present system as far as inspection of locally consumed meat is concerned, and advocating civic inspection of all meat sold for food. Prof. McDonald, Provincial Live Stock Commissioner, then gave a short account of what occurred at the recent convention of the Pacific Northwest Association of Dairy and Milk Inspectors, which he attended, being retiring President of that Association. Various questions were asked the different speakers and interesting discussion and information followed, bringing a very successful meeting to a close.

K. CHESTER, Secretary.



### VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

#### MINUTES OF THE MARCH MEETING

The regular monthly meeting of this association was called to order by the president, Dr. Goubeaud at 8:45 p. m.

The prosecuting committee reported progress and stated that they had about \$600.00 in hand.

Dr. McKinney mentioned the fact that Dr. Wertheimer had been arrested for practicing illegally and said that his health is poor. Stated that he intends taking the State Board and suggested that prosecution be held in abeyance.



Dr. Griessman stated that Dr. Wertheimer has had ample time in the past two years since his last prosecution, to qualify and suggested that he be placed under bond until he takes the State Board examination.

Dr. Goubeaud then gave a very complete and detailed report of the Trichiniasis cases which have recently occurred at Far Rockaway, New York. Stated that in one family four deaths had occurred and said that the convulsions preceding death were horrible to witness and the pain intense.

Was present with Dr. Silkman, chief veterinarian of the department of Health at the Post-mortem examination and the worm was found in the deltoid muscle.

It is stated that if the female trichinia is swallowed it will give birth to from 1300 to 1500.

Hogs affected with this disease seem to thrive and do not show physical symptoms.

Dr. Chas. S. Chase of Bay Shore, New York, then read an interesting and instructive paper entitled "Interesting Characteristics of Animal Parasites".

Among others the doctor mentioned the evolution of the Bot fly, also the Tsetse fly, which causes sleeping sickness. Also mentioned the peculiar condition of the *Filaria immitis* or so called heart worm which leaves the internal organs for the periphery at night. Cited a clinical case in a dog, which could be plainly seen under the lens.

Also gave the history of the development of the common house fly, stating that it is estimated that the progeny of a pair of flies in one season will reach into the trillions.

The most fatal enemy of the fly is an intestinal fungus which by the formation of gases causes the death of the fly.

To the common fly can be traced the spread of such diseases as typhoid, anthrax, leprosy, ophthalmia, small-pox, diarrhea in children and may be the cause of parasitic diseases. The doctor urges all veterinarians to use every means in their power to destroy flies.

Exhibited a fine specimen of *Filaria immitis* removed from the heart of a dog.

Dr. Gannett mentioned lice and said that it is stated that blue ointment rubbed in back of the ears once every five days will rid the animal of lice.

A general discussion of azoturia then took place.

Dr. Goubeaud opened the discussion by citing several cases of this disease.

One of these cases, affected in the nigh front limb, was given oil and turpentine—exercised the next day, developed the disease behind, went down and died.

Another case was found on arrival to be nearly dead. Gave physic, olive oil and turpentine, got up the next day and made a good recovery. A third and fourth case was treated with arecoline in solution and small doses given orally every two hours with good results.

Dr. McKinney stated that following the last Christmas holiday season he had fourteen cases of azoturia and had excellent results from the result of physic, oil and turpentine and chloral hydrate. In some of these cases bleeding was resorted to, and the animals given four grains of morphine and one grain of strychnine.

Dr. Schroder mentioned an odd case of a horse that was down in the street for three hours, received a strong electric shock from a trolley, got up and recovered.

Dr. De Vine said that he uses salt and avoids irritation by adding syrup or some other mucilaginous mixture.

Mentioned one case in which femoral paralysis resisted treatment for a long time, had a second attack, was down for three days, got up and paralysis was gone.

Dr. Wolters stated that in the case of paralysis following azoturia, it is best to work the animal as soon as possible.

Dr. Goubeaud said that it is best to turn them out to pasture and keep them out. This opinion was endorsed by a number of those present. Dr. Gannett mentioned one case that had paralysis of both hind limbs. Turned out for eight months and recovered.

The question of certain breeds being more subject to this disease than others was also mentioned. Percherons are said to be more subject to this bane of horse flesh than the Clydes.

No one present however claimed to have found a specific for this peculiar disease, so after a short general discussion the meeting adjourned.

ROBT. S. MACKELLAR, Secretary.

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The Connecticut Veterinary Medical Association will hold its summer meeting at the office of Dr. E. F. Schofield, at Bruce Park, Greenwich, Conn., July 25.

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VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

## MINUTES OF MEETING, MARCH 28, 1916.

A special meeting of this association was called by order of the president, Tuesday evening, March 28, 1916.

Dr. Goubeaud, who had just returned from Albany, explained that the object of the meeting was to devise ways and means of defeating the pending veterinary bills which had been introduced in both bodies of the state legislature.

Stated that in company with Drs. Berns, Ackerman, Way, Brotheridge, McKinney and Cochran, he had attended the hearing on these bills held by the Senate Committee.

Dr. Goubeaud said that the committee seemed convinced that this is special legislation and these bills should not have been introduced as the fact was brought out that one of these bills was introduced in the interest of J. L. Shorey of Schenectady, New York.

Owing to the possibility of these bills still being reported out of committee the following resolutions were unanimously adopted:

That this association goes on record as protesting against the passage of Senate Bill No. 537 now known as Senate Bill and Assembly Bill No. 709 now known as Assembly Bill No. 1041, and protest against the lowering of the standard of veterinary education and the requirement to practice veterinary medicine and surgery in the State of New York.

It was further resolved:

"That this resolution be referred to the Judiciary Committee directing them to draft a suitable letter to be printed, and sent to each member of this association, and all qualified veterinarians in New York City and vicinity, who in turn are to forward the same with a personal letter, to their representatives in the Senate and Assembly, or any others who are interested."

The secretary was also instructed to write Wm. A. Orr, secretary to the Governor, requesting him to notify the association at once should any of these bills be passed and come before the Governor for his signature.

Meeting adjourned:

ROBT. S. MACKELLAR, Secretary.

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F. S. Jones, V.M.D. of the Rockefeller Institute is to go to Patagonia, South America to investigate a disease among sheep.

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY  
MINUTES OF MEETING, APRIL 5, 1916.

The regular monthly meeting of the Veterinary Medical Association of New York City, was called to order by the President, Dr. Goubeaud, at 9 p. m.

Dr. D. H. Udall of the New York State Veterinary College had very kindly consented to make the journey from Ithaca, to address this meeting, and gave a very interesting and instructive talk on "Contagious Pleuro Pneumonia and Influenza."

The doctor described the conditions found in this disease from its inception. It is nearly always ushered in with a chill, temperature  $105^{\circ}$ , but sometimes a lower temperature is found. High pulse rate is always present.

The typical fever of lobar pneumonia is found.

Another characteristic is the high grade icterus of the membranes of the eye due to the breaking down of the blood and not to any disease of the liver. The lemon yellow exudate is also another peculiar symptom which is sometimes present.

Examination of the chest by striking a heavy hammer blow will immediately cause coughing.

Mortality ranges from four to twenty per cent.

Treatment depends largely on the condition of the heart.

The general treatment consists of the use of quinine, potassium, iodide, oil of camphor under the skin, camphor, caffeine, digitalis;—good hygienic quarters, fairly warm and airy but no draughts.

In the Prussian army, salvarsan has been used quite extensively.

In Germany strict quarantine regulations are enforced at the very first when any suspicion of the disease takes place.

Dr. Udall then took up the subject of influenza and said that it is transmitted very easily and the incubation period is only forty or fifty hours. One attack is supposed to produce immunity, but this is not always the case. It is one of the predisposing causes of pleuro-pneumonia.

This disease is found most often during the winter and spring.

When Dr. Udall had concluded his interesting and instructive address a number of the gentlemen took part in the discussion.

Dr. Gill said that Dr. Udall had covered the subject very thoroughly and speaking of pleuro-pneumonia said that in the cases brought to his attention, the left side was much more tender than the right and on post-mortem more lesions are found on the left side.



Said there ought to be some Federal Regulations regarding this disease to protect innocent purchasers as influenza and its complications cause great financial loss to horse owners every year.

Dr. Berns said that he distinctly remembered the so-called epizootic of 1873 and said that it was characterized by a copious discharge from the eyes and nose, accompanied by sore throat but the mortality was low.

Dr. McCully also gave a short concise report of his experience with influenza.

Dr. Ackerman spoke of the use of the biologic products in this disease but said that in his hands the results were not satisfactory.

Stated that recently he had drawn blood from a horse and used the attenuated serum to re-inject the same animal with the result that a full recovery resulted in a week. This same serum was used in other horses with good results.

Dr. Kingston stated that he has very good results from the intravenous injection of a 2½ to 5% solution of protargol. This it is stated will often give beneficial results and shorten the duration of the disease.

Dr. Ackerman asked in cases where digitalis is used if it does not cause or increase diarrhea. Dr. Gannet stated that in his experience it does not.

It was suggested that the attention of the proper authorities should be called to this disease and measures taken to prevent the great losses to horse owners. Crowded shipping conditions were also condemned.

A letter from Dr. J. G. Wills in which he called attention to Assembly Bill No. 1895 and urging this association and its members to oppose it was read.

It was on motion regularly made and seconded: That this association go on record as being opposed to the passage of Assembly Bill No. 1895 and that the secretary be instructed to write the chairman of the Assembly Public Health Committee informing him of the action of this association, unanimously carried.

The proposed amendments to the by-laws and code of ethics were then taken up.

Dr. Chase and others spoke against changing the date of the meetings of this association, and after a general discussion Dr. Gill moved that this resolution to amend the by-laws be referred back to the program committee for further consideration and to decide what is best for the interests of this association, seconded and carried.



The proposed amendment to the Code of Ethics making it a breach of the same for any member to employ a non-registered assistant, was after a rather lengthy discussion, not adopted.

Dr. Ackerman, chairman of the program committee, announced that at the May meeting Dr. Way had arranged to give an illustrated lecture, by the aid of moving pictures on "The Production and Handling of Clean Milk".

The secretary was instructed to invite the Commissioner of Health and other city officials as well as the heads of the different milk concerns and any others interested in this important work.

A rising vote of thanks was tendered Dr. Udall for his valuable contributions to the program of the evening.

Meeting adjourned:

ROBT. S. MACKELLAR, Secretary.

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## COMMUNICATIONS

*Editor of the Journal of the American Veterinary Medical Association, Ithaca, N. Y.*

Dear Sir:—

I have read with much interest and pleasure the valuable article of Dr. K. F. Meyer entitled: Studies to Diagnose a Fatal Disease of Cattle in the Mountainous Regions of California.

The discussions of Drs. Dunphy, Eichhorn and Simms (on this subject) are most excellent; but that of Dr. Kinsley is of especial value.

It seems evident from the heading and throughout the whole article of Dr. Meyer that some doubt has been experienced concerning the nature of the disease. I quite agree that the symptoms of hemorrhagic septicemia in some cases are not well pronounced and do not throw much light upon the nature of the disease and may even offer in several instances points which lead to a mistaken diagnosis; but this as a rule does not apply to all cases of hemorrhagic septicemia, on the contrary many cases are readily recognized and a clinical diagnosis is not impossible. I may further state that I have the opportunity of dealing with several outbreaks of hemorrhagic septicemia in cattle and Barbones in buffalos and believe to have had no difficulty in diagnosing the disease. I must confess though that in all the cases I have observed, none presented the intestinal form. In cattle the pectoral form was nearly always seen independently and in rare cases accompanied with the exanthematous form; in buffalos the exanthematous or edematous was the only form seen; in this form the local swelling of the throat is so intense and prominent that no difficulty is exercised in giving a prompt diagnosis.

It is, by the way, interesting to note that owing to the later swelling the Egyptian (fellah) farmer calls the disease "Khonag" which means strangulation; this seems to be quite an appropriate name; as it indicates the seat of trouble and helps diagnosis.

Now for the bacteriological examination, I personally believe that the presence of the *B. bovissepticus bi polaris* in the blood or tissue of the affected animals is quite diagnostic of hemorrhagic septicemia. It is useless to collect blood films for examination unless the animal is at the point of death, as the blood is only then virulent; therefore it is more advisable to collect films from the gelatinous substance generally found in the edematous swelling of the throat. The inoculation of this gelatinous substance is supposed to kill rabbits in from 8 to 12 hours; and the organism of hemorrhagic septicemia in blood of inoculated rabbits is so abundant that it could be easily detected even without staining.

Piot Bey\* seems to be quite convinced that the source of trouble in this affection is the watering of animals from stagnant water, and that is why he directed his attention and made some efforts towards digging wells near the bank of a canal, so as to get its water by filtration through a thick layer of earth or making some water pumps so as to allow the animals to drink pure and non-contaminated water. This way seems to put an end and stop the spread of the contagion. This has been tried for some years and seems to have answered quite satisfactorily in checking the further spread of the disease.

The mortality in cases of hemorrhagic septicemia in the locality where this way of watering was applied, amounts to 2-3%, while in places where this measure was neglected, the mortality went as high as 30 to 40%, bearing in mind that the sanitary police measures were properly carried through.

DR. J. E. AGHION,

Veterinary Inspector  
State Domains, Sakha, Egypt.

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## THE NECESSITY OF A LIVE STOCK SANITARY COMMISSION OR BUREAU IN THE STATE OF NEW YORK.

*Editor of the Journal of the A.V.M.A., Ithaca, N. Y.:*

Under Chapter 9, of the Laws of the State of New York, Consolidated Laws, we have the Agricultural Law. This law deals with the title and general provisions of the fifteen articles, only one article, No. 5, deals with Diseases of Domestic Animals.

The Commissioner of Agriculture is not a veterinarian, nor physician, but must seek advice on disease of our domesticated animals from a veterinarian.

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\*Literature consulted: Le Barbone du Bufile, par J. B. Piot Bey, Directeur du service veterinaire. Domaines de L'Etat. Extrait du Bulletin de l'Institute Egyptien. Année 1889.

A large responsibility is imposed upon the commissioner, in his duties to look after dairy products; adulterated vinegar; prevention of fraud in the sale of Paris green and other substances; sale and analysis of concentrated commercial feeding stuffs; sale, adulteration or misbranding of food and food products; sale and analysis of commercial fertilizers; turpentine, linseed or flaxseed oils; apples, pears, peaches, quinces; State Fairs, and other miscellaneous duties.

In justice to the commissioner, it is advisable to create this new commission or bureau, where the live stock problem of this state, could be handled and so regulated as to be of extreme benefit to the state and to the public.

Quoting from a bulletin of the United States Census, for the State of New York, I use their figures. In 1900, the value of live stock, New York State, was \$125,583,715; in 1910, the value was \$183,090,844, showing an increase of 45.8%. This increase is given as 44.7% of live stock during the same period, but the major percentage is 82.8% in poultry value and poultry stock.

We have a decrease in all cattle in this state, a total of 173,386 in ten years. There is a slight increase of 7986 dairy cows. A decrease of 37,430 horses in ten years, not including the amount of horses that have been exported during the past year and a half to the warring European nations; a decrease of 10,460 swine, and a very large decrease of 615,446 sheep.

There has been a general decrease in the live stock in this state as is shown, and, as I am given to understand, a very large decrease in all live stock from 1910 to 1915.

Under our present Agricultural Law, the owners of cattle and horses are indemnified for the slaughter of cattle suffering with tuberculosis, and horses infected with glanders.

From 1909 to 1915, 17,146 cows were slaughtered suffering with tuberculosis for which the State of New York paid \$812,944.43 in six years and during the same time 7221 horses were killed, diseased with glanders for which the state also paid \$494,524, making a total of \$1,307,468.43 in return for which these owners did not even contribute a single cent.

I propose and suggest a direct tax on the owners of live stock, as they are the only ones benefitting by the state indemnity.

We have, according to the figures below, the following live stock in this state and a small tax per head will bring an income into the state treasury of over \$3,000,000 a year.

Horses	591,008.	Proposed tax	\$1.00 each	\$ 591,008.00
Cattle	2,423,003.	Proposed tax	.50 each	1,211,501.50
Dairy Cows	1,509,594.	Proposed tax	.50 each	754,797.00
Swine	666,179.	Proposed tax	.25 each	166,544.75
Dogs	350,000.	Proposed tax	1.00 each	350,000.00
(my figures)				

\$3,073,851.25

This additional income to the state would defray the expense of this department and leave a sum of money approximately about \$2,000,000 to indemnify for the actual loss of tubercular cows, glandered horses and diseased swine.

I suggest that there be three directing veterinarians at a proposed salary of \$3500.00 and field veterinarians, one hundred or more at \$1800; experiment stations for the study of these diseases, state laboratory at the State Veterinary College for the examination of specimens, for the analysis of blood, the manufacture of immunizing serums, anti-toxins and viruses.

The Commission or Bureau should have the absolute right and power to control the suppression and eradication of contagious or infectious diseases of animals in this state, and to work in harmony with the animal division of the United States Department of Agriculture. The definite law or regulation as to the quarantine of animals; the compulsory testing of cows and horses for contagious or infectious diseases; regulating the shipping of live stock into and without the state and within the state from one county to another county; the issuing of live stock health certificates; that no horse, cow or pig could be sold without a health certificate, issued by the said bureau.

Inasmuch as the public has the benefit of the Pasteur treatment at the expense of the state, it is just that the owner of a dog shall pay to the state a tax of one dollar a year, and that the commission shall have the power to keep all dogs muzzled, so that rabies shall be entirely wiped out of the state of New York.

The directing veterinarians shall lecture on live stock, on the question of breeding, suppression of disease, sanitary conditions and good meat supply. The bureau or commission shall have the supervision of the local slaughtering houses for the inspection of meat.

Employment of veterinarians shall be from the state civil service list, and all veterinarians required to report once a year in writing as to their post office addresses.

Bulletins shall be sent out at such times to all veterinarians, informing them of any change in rules or regulations.

Veterinarians shall have control as to the testing of live stock and in the event of their failure to make true and accurate tests and reports, to have the right to have the individual veterinarians' licenses revoked.

It shall be a misdemeanor for anyone other than a licensed or registered veterinarian to inject any tuberculin, mallein or virus or toxin for the purpose of establishing the fact that said animal is suffering from any disease.

The various health departments or bureaus in the different cities or towns, having veterinarians in their employ shall come under the supervision of the said commission or bureau, and they shall do the work as if in the employ of this commission or bureau,



and the head of that division shall make weekly reports to this commission.

The reason for the above is that it shall not be necessary for two departments to do one act, and the state shall not be encumbered with the payment of a salary for unnecessary work that is and had been performed by the local health bureaus.

The control and shipment of the manufacture of antitoxins, vaccines, or any serums shall come under the supervision of this department, that is, where they are manufactured within the state, or that the standard of any such vaccines or antitoxins must come up to the standard of the United States Bureau of Animal Industry.

Any fake remedy for the so-called cure of diseases in animals shall be under control.

There shall be compensation to veterinarians for the reporting of any contagious or infectious diseases.

LOUIS GRIESSMAN

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*Editor of the Journal of the American Veterinary Medical Association, Ithaca, N. Y.*

Enclosed herewith please find my personal check for two dollars (\$2.00) with which to be the nucleus and start a fund for the expense necessary to have moving pictures made for the A.V. M.A. and subsequent state meetings, of the various operations by expert veterinarians as indicated in the close of the paper which I gave at the Missouri Valley Veterinary Medical Association in July, 1914, entitled "The Sturdy Farm Boy, etc."

I note that the idea was successfully carried out at the American Medical Association and the veterinary profession ought to be able to do the same.

BURTON R. ROGERS.

[EDITOR'S NOTE:—Dr. Rogers' communication reached us just before the later forms of the *Journal* went to press. In order that the idea may not be delayed we are publishing his letter. If other donations are received they will be held until some definite plan is arranged. If the plan does not materialize the donations will be returned to the senders.]

The mid-summer meeting of the Illinois State Veterinary Medical Association will be held at Peoria, Ill., July 19th. The officers are F. H. Burt, president; A. W. Smith, vice-president; L. A. Merillat, secretary-treasurer. The annual meeting will be held at Chicago, Ill., December 5, 6, and 7.

The next meeting of the Montana Veterinary Medical Association will convene in the Capitol Building, Helena, September 27 and 28.



## REVIEWS

### VETERINARY THERAPEUTICS

E. WALLIS HOARE  
Alex. Eger, Publisher, Chicago, Ill.

This book is now in the third edition. It contains 943 pages and covers the greater part of the entire subject of Veterinary Therapeutics. The text is divided into three parts.

Part. I. This contains chapters on the general diagnosis of diseases; care, management, and nursing of sick animals; actions and uses of drugs and veterinary pharmacy.

The chapter on general diagnosis of diseases treats of the usual physical examination, examination of the urine and an interpretation of various symptoms of disease. The ground is covered quite thoroughly but of course is not so complete as the usual texts on the various subjects.

The chapter on the care, management and nursing of sick animals includes specific directions for each group of patients, including birds. It is composed of several articles by different men who are evidently specialists on the various subjects. Thus, Hoare is probably the author of that part pertaining to horses, Hugh Begg, for cattle; Henry for dogs, cats and birds and G. Mayall for sheep and swine. These articles are all very complete and contain a vast amount of information not usually found in such a book, although there is considerable material which is foreign to the subject.

The sections on the actions and uses of drugs includes discussions on the general effect of drugs, methods of administration and a brief discussion of the different classes of drugs grouped according to their action. The last part is very brief and amounts to but little more than definitions and examples in many cases.

Part II. *Materia Medica*. This is really pharmacology and general therapeutics. It discusses the physiological actions and the uses of the individual remedies which are of importance in veterinary medicine. The author has included, not only most of the official and more popular drugs, but many proprietary and semi-proprietary remedies. The subject matter is fairly complete, but the grouping is such in many cases as to render it difficult as a text.

Part III. This part is devoted to the treatment of those diseases most commonly found in veterinary practice, minor surgical operations and a formulary of prescriptions for various ailments.

The final chapter on vaccine therapy is written by W. M. Scott and includes a valuable discussion on immunity together with articles on the more commonly used immunizing agents. The diseases are grouped under the general headings: diseases of the digestive system, etc., and sub-headings devoted to the different species of animals, horses, cattle, dogs, etc. The greatest emphasis is based upon the treatment of the various diseased conditions, but, in many cases, the causes and symptoms of the disorders are briefly reviewed.

The minor surgical operations include such common operations as firing, passing the probang and stomach tube, puncturing the intestines and rumen, and catheterization. Very exact directions are given for these procedures, but the reviewer is not competent to give an opinion of them. It seems, however, that the average practitioner, should already be familiar with most of the operations described and this text cannot in any way take the place of several texts on surgery.

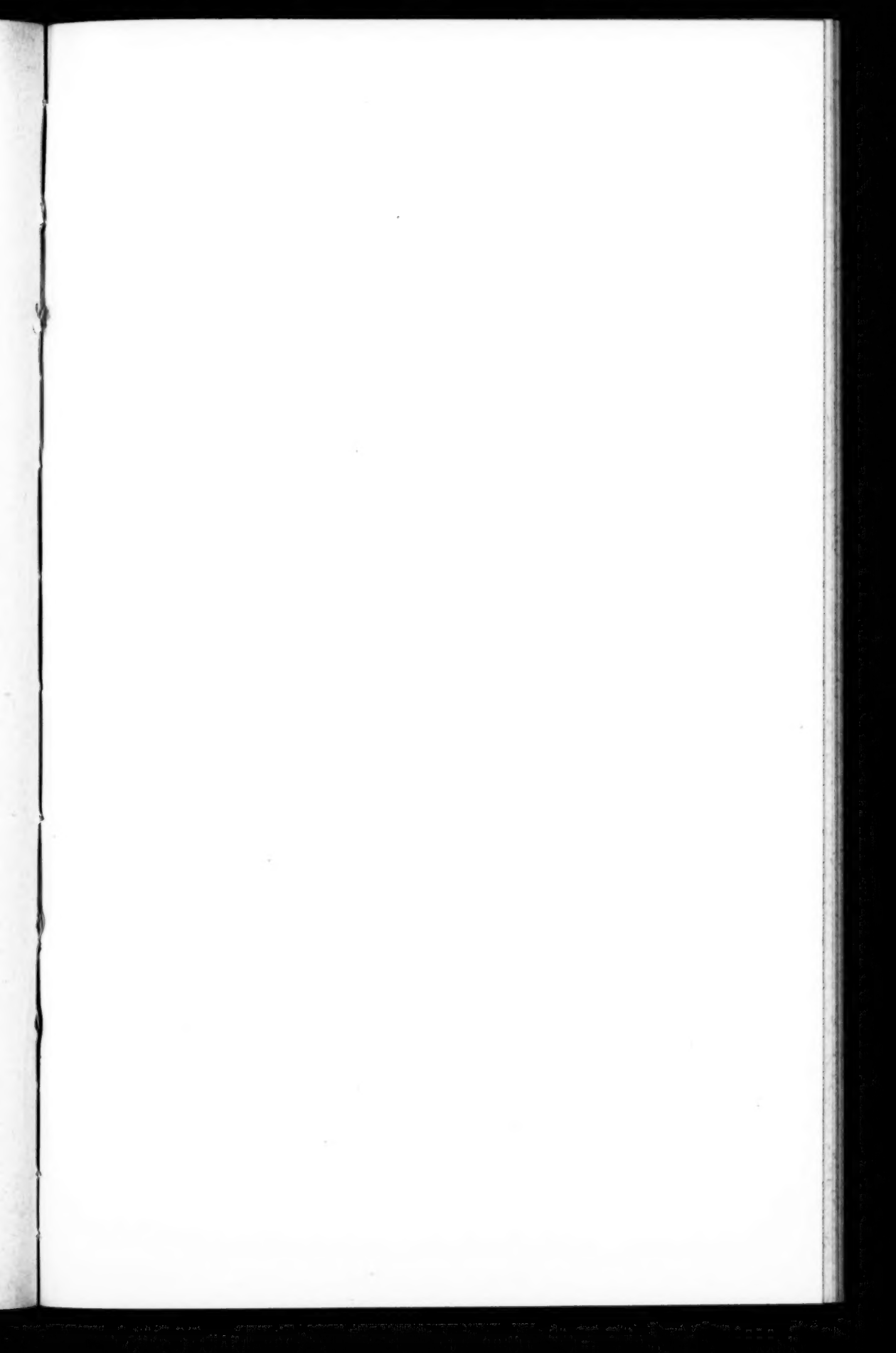
Finally, there is an appendix of some 40 pages of formulae for various conditions.

Veterinary Therapeutics is evidently a valuable addition to veterinary literature but, in the opinion of the reviewer, attempts to cover too briefly many subjects which are more thoroughly discussed in special texts on the subjects, and with which practicing veterinarians should already be fairly familiar. Furthermore, it does not appear that it can take the place of our texts on physical diagnosis, urine analysis, pathology and surgery, so that certain parts although exceedingly valuable should not be utilized as text books.

This book should prove valuable to any practitioner, although unfortunately the subject matter and prescriptions are based upon the British Pharmacopoeia instead of that of the United States, a fact that will tend to cause confusion to American Practitioners or pharmacists.

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The Maine Veterinary Board of Examiners met for the purpose of organization in Augusta on June 14. Dr. I. L. Salley of Skowhegan was elected president. Dr. W. H. Lynch of Portland was elected secretary, Dr. W. H. Robinson of Woodfords, treasurer. A meeting of this board for the reception of candidates will be held in the last week of July at Augusta.





**R. A. ARCHIBALD**

**President of the American Veterinary Medical Association  
1915-1916**